THE BORDER-LAND OF EPILEPSY

FAINTS, VAGAL ATTACKS, VERTIGO, MIGRAINE, SLEEP SYMPTOMS, AND THEIR TREATMENT

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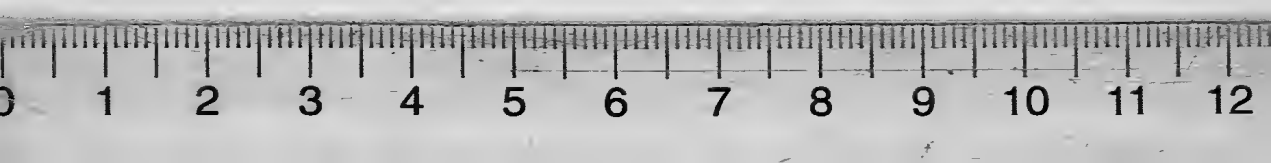

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PREFACE.

FOR many years I have kept a special list of all cases which seemed to be in the border-land of epilepsy—near it, but not of it. Many were so placed by their features and character; others because they had given rise to an erroneous diagnosis. When these cases were collected and classified, their comparison and study revealed a large number of unfamiliar facts and many instructive lessons, throwing light on the nature of the affections, on their relation to epilepsy, and on questions of practical diagnosis. The most important of these facts and conclusions are given in the following pages. Although most of their substance has appeared, as lectures, in the 'Lancet' and 'British Medical Journal,' the importance of the facts seemed to justify their collection in a more permanent form. For this purpose the following chapters have been largely re-written. An account of some sleep symptoms has been added, and to the description of each border-land malady a section on treatment has been appended.

The unfamiliarity, to most persons, of many of the facts and conclusions made it desirable to describe the evidence on which they rest. This has been done as briefly as possible. Although only the essential details of the cases

are given, they may make a hasty perusal less easy. But the method has the advantage of bringing the facts more vividly before the careful reader, and of aiding their retention in the memory. It also facilitates their use when a need arises for their practical application. For the most part they will be found to have a wider importance than their mere relation to epilepsy would suggest.

W. R. GOWERS.

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THE BORDER-LAND OF EPILEPSY.

CHAPTER I.

FAINTS AND FAINTING.

A GENUINE "faint" bears a close superficial resemblance to an attack of minor epilepsy, and hence cardiac syncope is a convenient starting-place for a survey of the borderland of epilepsy. The resemblance often entails a difficulty in diagnosis, and even involves problems regarding this nature of the two conditions, a consideration of which is instructive.

Familiar as fainting is, adequately as we seem to know it, there is much in it that we do not know. Our knowledge is enough to obscure our ignorance. The most obtrusive feature of complete cardiac syncope is the loss of consciousness which results, evidently due to the failure of the action of the heart which precedes and attends it. But the loss cannot be the direct effect of the cardiac failure, because consciousness is not the result of the circulation of the blood. To say this is to state an obvious truism, for the two are totally different in nature. The loss must be immediately due to a state of the nerve elements of the brain produced by the change in the circulation. We are apt to overlook this when we think of the process of fainting, but its recognition is of great

importance because consciousness may be lost from other causes. It is the most common feature of the epileptic seizure. Not long ago it was thought to be a constant feature; without such loss an attack was said not to be epileptic. We now know that minor attacks are common in which consciousness is only dimmed; sometimes hardly a ruffle on its surface attends the sensation which constitutes the slightest form of attack. Still, in the pronounced form of each condition, in fainting and in epilepsy, loss of consciousness is a dominant feature. The fact is of practical importance in diagnosis, because it often makes their distinction difficult, and sometimes causes a mistake. For another reason, also, the loss of consciousness is important. We do not know in either malady the nature of the process in the nerve elements on which the symptom depends. We do not know whether it is the same in the two or is different. If we can trace any relation between the affections it will constitute some evidence on the question, evidence at least suggestive. For this reason also the study of fainting in relation to epilepsy is important.

Recognising that a change in the nerve elements underlying the loss of consciousness results from the failure of the circulation, another question presents itself. How is this nerve change brought about? Perhaps the first explanation that would occur is that the heart's failure entails a diminution in the supply of nutrition to the brain, incompatible with the maintenance of its highest functions. A little consideration will make us doubt the adequacy of this mechanism. The renewal of nutrition of the nerve elements, the supply on which their metabolic processes depend, is from the plasma about them, derived from the blood, but for the time extra-vascular. At any moment the amount of this must be adequate to maintain the metabolic changes, and the function that depends on

these, for a longer time than that during which consciousness is maintained in syncope. How inadequate this influence must be to account for the loss is evident from the features of the fatal syncope of aortic regurgitation. The sudden death in this disease, rare though it is, is clearly syncopal, and almost synchronous with the cardiac failure.

Another mechanism, far more adequate, consists in the mechanical effect of cardiac failure. A constant pressure is exerted by the force of the blood in the arteries of the brain, a pressure which must be considerable, and to it the brain is habitually exposed. If the heart fails the resulting diminution in the pressure within the cavity of the skull must be great. It will be effective in proportion to its suddenness, as are all variations of intra-cranial pressure. The case last mentioned, the sudden arrest of the heart's action in aortic regurgitation, must be mechanically equivalent to a "stunning" blow. In the more gentle failure of ordinary syncope the mere diminution in pressure is conceivably adequate to induce the alteration in the nerve elements on which unconsciousness depends, whatever its nature.

We do not know what this process is, and it is not easy to frame an hypothesis in harmony with its varied causation that will permit it to be regarded as the same in all cases. We have to conceive it as occurring spontaneously in epilepsy, and as due to a sudden diminution in the intra-cranial pressure in syncope, acting as a mechanical influence, and it may also be due to a sudden increase in the pressure exerted by the blood. The effect of a sudden blow on the skull, which abolishes consciousness without causing any visible lesion, is another example of a mechanical cause having the same effect.

Only one hypothesis agrees with all the causes, and that

is, perhaps, incapable of proof. Many phenomena in the nervous system can only be intelligibly explained on the assumption of a discontinuity of conduction at the junction of the neurons, which compose each conducting path. At such interruptions impulses are re-excited, instead of simply passing on. It is held by many authorities that this discontinuity is at the terminations of the branching processes of the nerve cells, the "dendrites" as they are termed. It is supposed that these structures possess some mobility, some power of retraction and elongation, by which their distance from the exciting structures can be varied. A movement extremely slight may suffice to arrest the action of one set of structures on another. It may, for instance, absolutely disconnect from all lower centres those that subserve consciousness. The hypothesis is still unproved, but it explains much that we cannot otherwise understand.* It enables us to conceive that the mechanism may be the same which causes loss of consciousness in both fainting and epilepsy. In the latter the process must occur through a wide region with almost absolute suddenness, and we can conceive that such activity may spread with explosive rapidity. We can also understand that a sudden mechanical influence may induce the same change in structures that may reasonably be thought to possess most delicate susceptibility. On this hypothesis only can we conceive that the same nerve process may underlie the loss of consciousness in both fainting and epilepsy.

We have to think under the limitations of present knowledge. What change in thought the future may have in store for us we know not. "The new knowledge" holds before us the prospect that all material elements will be discerned to be forms of electrical energy, and atomic combinations will resolve themselves into electrical flux and

* See "Dendrites and Disease," 'Lancet,' 1906.

change. If so, organic chemistry must share the altered thought, and the mysterious electrical changes that attend nerve action may thus become intelligible. What explanation such facts may afford of the phenomena of arrest and resistance in the central nervous system the future alone can show. At present we cannot discern grounds for framing any other hypothesis to explain the phenomena than that just stated.

To resume the consideration of the facts of disease. It is doubtful whether true cardiac syncope ever causes absolutely sudden loss of consciousness, except when this is due to a fatal arrest of the action of the heart. It is seldom, if ever, so sudden as to cause a hurtful fall. As a rule, the deliberate onset enables the sufferer to lie down, when gravitation ceases to hinder the flow of blood to the brain. The return of strength to the pulse and of colour to the face is attended by deeper breathing. Gradually consciousness returns, and with it correct perception of surroundings, never the mental confusion and erroneous ideas or action that are common after minor epilepsy. On the other hand, after the latter there may be an instant return of a normal mental state, with no transitional sense of lessening prostration that is the usual mode of recovery from syncope. Moreover, it is important to emphasise the fact that in minor epilepsy, which alone can be confounded with cardiac syncope, there is never initial pallor of the face. This follows the onset. Error on this point is common because the patient is only closely observed after some feature of the attack has drawn attention to him. Much more frequently no change in the tint of the face is to be seen either before or after a minor seizure. This is true also of the pulse. The state of the vessels of the retina does not necessarily indicate a similar condition in those of the brain, but I once chanced to

have those of the retina under clear view by the direct method of observation throughout a minor attack, and no change in them could be seen.

Causes and Mechanism of Syncope.—We shall see better how imperfect is our knowledge of syncope if we consider the various causes by which the failure of the heart may apparently be brought about. One obtrusive cause is loss of blood, and another, similar in nature, is the withdrawal of a large amount of blood from the general circulation by its collection in one set of vessels. You know that fainting may occur when much fluid is withdrawn from the peritoneal cavity, unless the abdominal vessels are supported by external pressure. This is, no doubt, the explanation of one of the commonest causes of fainting in strong men, violent diarrhœa or a violent purging. The flux to the intestinal vessels, and the copious efflux from them, render the amount of blood in the general circulation inadequate. The effect of a hot room is ascribed to a similar mechanism, the dilatation of the vessels of the surface, although in this case other influences may perhaps cooperate.

In these cases how, we may ask, is the failure of the heart brought about? Is it produced by the diminished flow in the arteries of the heart, or by the lessened amount of blood within the cavities being insufficient to excite the contractions? Or does the diminution in the blood-supply to the medulla oblongata excite an impulse in the pneumogastric which causes the heart to fail? The last involves two questions. Is the inhibitory influence from the cardiac centre in the medulla excited in it primarily by the change in the circulation, or is it due to the influence of this change on the cerebral hemisphere, the effect being focussed down on the medulla?

Syncope has other causes which concern more directly our present subject. It may be produced by an influence that acts directly on the nervous system and seems to act through it. One such cause is a sudden intense pain, especially if felt in the abdomen or in the vicinity of the heart. The mechanism is supposed to be a direct action on the centre for the vagus, but syncope is only known to result if the pain is perceived. In man, I think, a cause of adequate pain has not been known to produce syncope if the patient was under the influence of an anæsthetic. Hence it seems doubtful whether the effect is due to a direct action on the vagal centre; the facts suggest that it may be the result of a profound influence on the sensory regions of the cortex, focussed down on the cardiac centre in the medulla.

The disturbance of the cortex must also be concentrated on the cardiac centre when syncope results from sudden intense emotion, which is a well-known cause. It may result whether the emotion is one of fear, distress, or joy. As the French saying states: "La joie fait peur." The action on the centre for the heart must then be through the cerebral cortex. There is no reason for the assumption that emotion acts by causing vaso-motor dilatation elsewhere. No evidence of such an effect can be discerned, much less on a scale adequate to produce syncope, while an influence of emotion on the vaso-motor centres is not easier to understand than is one on the cardiac centre.

The syncope that may be caused by the sight of blood is also well known. This must be purely emotional in its production. It does not seem to be caused by depicted blood, however realistic may be the painting. A special idiosyncrasy renders some persons liable to faint when certain odours are perceived, especially those of certain flowers, even such as are purely pleasant to most persons.

By what mechanism the result is produced is unknown. It is difficult to conceive that the stimulation of the olfactory nerve has a direct effect on the vagus. It seems necessary for the odour to be perceived; so that here again the influence on the cerebral cortex may be necessary for the effect.

Syncope and Minor Epilepsy.—It has been mentioned that the absence of evidence as to the actual nature of the process that underlies loss of consciousness in the two conditions, gives much importance to any indications of a connection between syncope and epilepsy, to any cases in which repeated syncope seems to pass into minor epilepsy. Such cases constitute evidence that the state of the nerve elements is the same in the two conditions, and possess, moreover, considerable practical importance. They are rare, but the relation seems to be presented by the following cases.

A girl, when about seven years old, became liable to faint on any sudden start or alarm. The faints had all the characters of cardiac syncope; there were pallor, coldness, and gradual loss of consciousness. After some years such pains occurred without any sudden excitant and became more sudden in onset. When she was seventeen years old one of these attacks passed into a distinct epileptic fit, with deviation of the head, general clonic spasm, and micturition during the attack. Others followed.

A single woman, aged 34 years, had been liable for many years (twelve or more) to apparently true fainting attacks, caused by excitement, over-fatigue, or a hot room. They began with palpitation of the heart and a sense of faintness, followed by gradual loss of consciousness, which did not become complete if she was able promptly to lie down, when she always began to recover. At the age of

twenty-five years these faints changed in character. She became unconscious more suddenly and without any exciting cause; indeed, the onset was so abrupt that she often only knew she had had an attack by finding herself on the floor. Recovery was then by mental confusion, not lessening faintness, and at first she could not tell where she was.

Another patient, regarding whom the facts could be ascertained with considerable precision, was a woman who became prone to faint in early girlhood, especially in hot, crowded places. In these she first became pale, felt faint, and gradually lost her sight, and then, if she tried to stay where she was, she lost consciousness. Usually, when sight had failed, with help she was able to walk out of the place. During the effort dim sight returned and the fresh air quickly restored her. These are the typical symptoms of cardiac syncope. Such faints continued until she was thirty years of age, when she became liable to sudden brief loss of consciousness, in which she often fell and sometimes hurt herself. After these she usually slept and could not afterwards recall what had happened between the attack and the sleep. One occurred in a room with the door locked; the noise of her fall alarmed her friends, who knocked at the door. She got up and opened it, then she lay down and went to sleep, but afterwards remembered nothing of the incident. Such attacks were undoubtedly epileptic, and after they began she ceased to be liable to the faints. She never had a convulsive seizure, but the attacks continued until she came under treatment at forty-two years of age, when they were arrested by bromide; she has since continued free. There was no heart disease and no family history of epilepsy, and no cause could be ascertained except the apparently disposing influence of the cardiac faints.

These cases present strong evidence of the influence of

repeated cardiac syncope in disposing to epilepsy. They suggest that the state of the nerve elements that underlies the loss of consciousness in syncope may, by repeated induction, acquire a tendency to spontaneous development, which constitutes minor epilepsy.

It should be noted, moreover, that those who are prone to faint, become, after a time, liable to do so more suddenly. A woman of fifty had always fainted readily, under such causes as over-exertion, etc. The faint was always preceded by a sensation at the back of the neck, and when she felt this she laid down and gradually recovered. But once she felt this sensation and knew nothing more until she found herself on the ground and her face being bathed. She had lost consciousness completely, with only the briefest initial sensation. The fact suggests that the brain-process underlying unconsciousness had here also acquired a special tendency to instant development.

Attacks of unconsciousness occurred with the suddenness of minor epilepsy, after an excitant such as induces syncope, in another patient. She was a married woman, and at thirty-eight she came under observation on account of slight hemichorea of two months' duration. She had had an attack of chorea at nineteen years of age, lasting for two months, and another at thirty-three years of age, lasting for six months. There was no heart disease. At the age of thirty-four years she cut her finger badly; two days later the bandage suddenly slipped off and at the sight of the cut she instantly fell unconscious, hurting her nose in the fall. She breathed heavily for ten minutes and then recovered. When she was thirty-seven years of age she again cut her finger slightly and immediately became "dazed," became pale, and then lost consciousness for five minutes. The first of these attacks had the definite features of minor epilepsy in suddenness and

character, although excited by an occasional cause of syncope. The second was due to a similar cause, and resembled syncope in its less sudden onset and in the fact that pallor preceded loss of consciousness, but before the pallor there was definite disturbance of function in the brain.

The features of these cases are of obvious significance in relation to the question of the identity in nature of the cerebral process.

Diagnostic Difficulty.—Whatever may be the nature of the process underlying the loss of consciousness in syncope and in minor epilepsy, the symptom sometimes brings the two affections near together in the practical difficulty of distinguishing one from the other. This is sufficiently frequent to be of considerable practical importance, and its study is instructive in many ways, revealing facts that might otherwise escape attention. Sometimes the difficulty exists when it should not, because the symptoms are sufficiently distinctive. That it may be reasonable is evident from the fact that attacks which are originally syncopal seem sometimes to become epileptic. But when the diagnostic difficulty is not reasonable, it may be very real.

The error of regarding minor epilepsy as cardiac faints, which is the more common, is often promoted by the natural tendency to perceive that which is less grave. The distress caused by the recognition of epilepsy is always great, and naturally exerts a deterrent influence. A pertinent example recently came to my notice, which conveys a special lesson in diagnosis. A patient with no relevant disease told me, as an unimportant incident, that he had recently fainted just as he had finished his dinner. He had taken nothing that was unwise. On inquiry as to

the features of the faint, he said that just as he had finished the meal, before rising from his chair, he suddenly felt extremely sleepy and knew no more until he heard the words, "Are you better now?" from a companion, who, seeing he was unwell, had saved him from falling off his chair. He had been unconscious about two minutes, becoming dusky in aspect. On recovering he was well almost at once. These are not the features of a cardiac faint. Such sudden, intense, brief sleepiness is not met with at the onset of syncope and is often met with in minor epilepsy; this is true also of the dusky tint, which indicates impairment of breathing, not of the heart, while the very sudden return to the normal state is also an epileptic indication. Whatever were its immediate cause and remote significance, it was certainly not a cardiac faint.

Attacks are sometimes thought to be syncopal when their features indicate that they are independent of the heart. This may be promoted, not only by a tendency to take the milder view, but also by the fact that sometimes their features may suggest a cardiac origin. One of these is a sensation referred to the heart or its region. When it is really an epileptic aura, it may seem to indicate that disturbance of the heart is the first element, and that its failure causes the loss of consciousness. Such an error is the more easy because a cardiac aura is not common in epilepsy, and its occurrence may even be unknown. Such ignorance was clearly the cause of the mistake in a boy, aged 16 years, who had "fainted" about a dozen times during a fortnight, nine months before he was seen, but not again until the previous week. During this he had fainted eight times, always in the day. In the first series, each faint had begun by a sudden stab at the heart, instantly followed by unconsciousness. In the later attacks the cardiac pain was also the first

symptom, but was slighter and less brief and was accompanied by a sense of nausea and giddiness. After a few seconds he fell from sudden loss of consciousness. On its return, after a "minute," he struggled and had to be held down, evidently in the hysteroid state that so often follows minor epilepsy, but never occurs after cardiac syncope. Yet the case had been regarded as one of simple cardiac faints on account of the initial pain, which was really an epileptic aura. It is noteworthy that when it became more deliberate the disturbance spread in the central relations of the vagus, causing the sense of nausea and associated giddiness.

Less frequently, simple cardiac faints are thought to be minor epilepsy. The mistake is especially easy when the patient is a member of a family in which epilepsy has occurred. Heredity naturally excites suspicion as to the nature of apparent faints. It was so in the case of a girl, aged 18 years, whose cousin was epileptic. Brief loss of consciousness had occurred several times during the preceding year, and the attacks were regarded as minor epilepsy. But each had occurred under conditions capable of causing cardiac syncope. Each began with pallor and a sense of faintness; in each sight failed first, and then consciousness was gradually lost. There was no rigidity, and recovery, on being laid down, was by a slowly-lessening sense of faintness, without any mental confusion. The opinion that they were simple cardiac faints was confirmed by complete recovery under tonics.

As the last case shows, the fact that an attack is excited by an external influence is important as confirmation of its syncopal nature, when this is also indicated by its features. But too much weight should not be placed on the fact that it is thus induced, unless the exciting influence is distinctly such as causes fainting, and the character of the attack is that of cardiac syncope. As a general rule, in

the differential diagnosis of disease symptoms must be combined and their relative weight considered.

Epileptic attacks are sometimes excited by an external influence, even by such as might seem likely to cause syncope. A sudden noise, such as the slamming-to of a door, may cause an attack of minor epilepsy in those who are subject to it. A sudden sound is always, as we say, "startling." The "start" is due to a sudden stimulation of the motor centres, which represents the instant motion to escape from danger, which is so familiar to us in lower creatures, and has, perhaps, produced our general term "e-motion." The wide and instant effect on the cortical centres renders it easy to understand that it should sometimes excite an epileptic seizure in those who are liable; indeed, we may feel surprise that the effect is so seldom met with. A sudden alarm occasionally induces a true faint, but the alarm is usually more profound than that caused by a sudden sound, and the syncopal attack comes on deliberately.

Sudden change of posture is an occasional excitant of loss of consciousness. If the change is to the erect posture it may be regarded as evidence that this is syncopal. For the moment the flow of blood to the head must be hindered. But in some cases of undoubted epilepsy, the movement may induce an attack. I had once an opportunity of observing the effect. Consciousness was instantly lost; the patient became rigid and would have fallen unless supported; the conjunctival and pupillary reflexes were absolutely lost. Had it been syncope the onset would have been deliberate, preceded by pallor, and there would have been no rigidity. The mechanism of an epileptic attack thus induced is an interesting problem. The facts suggest the possibility that the sudden diminution in blood-pressure, trifling as it must have been, may have caused the nerve-elements to pass into the condition

which underlies the unconsciousness of a complete syncopal faint. They again bring before us the problem of the identity of their condition.

Stooping, lowering the head, has the opposite effect on the intra-cranial circulation; it tends to increase the amount of blood in the vessels, especially when combined with flexion of the neck (hindering the return by the veins) and with some effort, which is usually the object of the stoop. The fulness of the vessels thus induced may sometimes cause a momentary loss of consciousness. A more intense mechanical congestion is induced by severe paroxysms of cough, such as those of whooping-cough. They may culminate in a moment's unconsciousness, as if the sudden increase of blood-pressure produced the same functional state of the nerve elements as its sudden decrease. We must remember that the mechanical effect of a sudden change of pressure may have the same functional result, whether the sudden change is an increase or a decrease.

The fact is illustrated in an instructive manner by the case of a woman, aged 38 years, who had epileptic attacks in the night, apparently of minor character, but attended by micturition. They also occurred in the day, and then began with a sensation of a sudden rush to the head, followed by loss of sight and then loss of consciousness. Automatic action followed the attack. One was so sudden in onset that she fell unconscious on the floor. Thus there can be no question of their epileptic nature. In her the act of stooping often induced a partial attack; there was a sudden rush to the head, and complete loss of sight, but there was not actual unconsciousness. Sight gradually returned on rising. Otherwise these attacks, induced by stooping, resembled closely those that were spontaneous. The lesson of the case is that the increased intra-cranial pressure produced by lowering of the head excited a change in the nerve elements similar to that

which underlies the epileptic seizure. Moreover, the effect bore some resemblance to the features of cardiac syncope, but was more rapid in its course. It is in harmony with other facts that either increase or diminution of intra-cranial pressure, if rapidly produced, should have a similar result on the cerebral functions.

These facts again bring before us the probability of the essential identity in nature of the process in the nerve elements underlying the loss of consciousness in the two conditions. The hypothesis I have mentioned enables us to conceive that the repeated induction of the process by sudden diminution in the blood-pressure may sometimes produce a tendency to its spontaneous occurrence, spontaneous at least as it seems to us. In less degree the same facilitation may enable a slight influence to produce a similar effect. We know that in the nerve centres repetition promotes the recurrence of any form of activity, normal or morbid.

Treatment.—The repeated occurrence of fainting seems to facilitate the process, and even to render easier the production of unconsciousness. Moreover, some persons faint more readily than others. Hence, it is important for those who are prone to pass into the condition to avoid the influences that tend to bring it on, even at some sacrifice of pleasure. All strain of body or mind is harmful, and physical exertion in hot surroundings is especially prone to induce physical collapse. All hot and crowded rooms should be shunned, but gentle exercise in the open air ought to be made a regular part of life, as a duty, in which women too often fail.

All digestive derangement should be met by appropriate treatment; flatulent distension of the stomach is especially apt to disturb the heart and to make a slight excitant

effective in causing fainting. Exertion after meals, especially such as involves stooping, should be carefully avoided. Clifford Allbutt has noted that in those in whom change of posture has an undue effect on the cardiac frequency, exceeding five beats per minute, greater care than usual is needed.

Most sufferers need tonic treatment. A tendency to faint often depends on general debility, to be met by iron if there is anæmia, sometimes combined with arsenic, and in other cases by quinine, strychnine, etc. In cases of pronounced nerve-weakness free phosphorus is often very useful; its value is more distinct than that of the compounds so largely used, the utility of which seems chiefly due to the combined bases. In all cases with cardiac feebleness, whether shown by frequency, weakness of pulse and sounds, or irregularity, heart tonics are needed, digitalis or strophanthus.

Whenever there is evidence that loss of consciousness occurs with undue rapidity, sooner and more quickly than in the ordinary course of syncope, the epileptic aspect of the seeming faint should be noted. This is increased by sudden return of consciousness, or by its return through mental confusion rather than physical faintness. In these cases bromide may wisely be given, at any rate if such attacks are repeated, or tend to occur after an inadequate excitant. But a single attack of this character does not justify a diagnosis of epilepsy, with all its attendant concern and anxiety.

CHAPTER II.

VAGAL AND VASO-VAGAL ATTACKS.

THE term "vagal attacks" is unfamiliar, but it seems a useful designation for prolonged seizures, the symptoms of which consist chiefly in disturbance of some of the functions of the pneumogastric. They are for the most part sensory and therefore subjective. The word "vagal" is used only as a convenient descriptive term, bringing together symptoms which cannot easily be included in a useful designation. The symptoms comprehend subjective gastric, respiratory and cardiac discomfort, sometimes cardiac pain and even a sense of impending death. With the vagal symptoms there are often combined a slight mental change, and also disturbance of the vaso-motor centre, causing constriction of the vessels and coldness, especially of the extremities. Associated with the latter may be some sensory impairment and often also a form of slight tetanoid spasm. These features vary much in relative proportion, so as often to obscure the essential resemblance. When the vaso-motor spasm preponderates, the case may seem to differ from the type more than it really does. Such cases may be termed "vaso-vagal." The attacks are never really brief; they seldom last less than ten minutes and more often continue for half an hour or more. There is a sudden onset of slight symptoms, rapidly increasing, and the ending is

gradual. The seizures recur at varying intervals, often for months or years.

Cases of allied character have received some attention in connection with the resemblance to angina pectoris, when cardiac distress and pain are prominent and are associated with vaso-motor symptoms. They were described by Nothnagel* as "angina vaso-motoria" and also by Sir Douglas Powell,† and some of the symptoms were mentioned by Bonnier‡ as "syndromes medullaires." But none of these writers have brought the symptoms together as features of definite attacks, irrespective of their relation to the heart.

Although the term "vagal" is used as a descriptive term, without implying causation, it may be remembered that the vagus is the nerve through which respiratory and gastric sensations are felt. The experiments of Dr. A. Waller on compression of the vagus are worth recalling. He found that pressure on the nerve behind the carotid in the neck caused first a sense of want of breath, followed by deep and laboured inspiration, uneasiness over the precordial region, and discomfort in the stomach, sometimes amounting to nausea. If the pressure was maintained, syncope was the result. He satisfied himself that the symptoms were not due to the compression of the carotid.§

Women suffer more frequently, but these attacks are

* Nothnagel, 'Deut. Arch. f. klin. Med.,' Bd. iii, p. 309; Nothnagel (in the same, Bd. ii) described vaso-motor spasm in the limbs, causing coldness and tingling in the extremities.

† Allbutt's 'Syst. of Med.,' vol. v, Art. "Angina Pectoris."

‡ Bonnier, 'Le Vertige,' Paris, 1904. Some of the symptoms have been described in other associations, by Freud, Kartenberg, and others, especially in connection with "neurasthenia."

§ A. Waller, 'Proc. Roy. Soc.,' vol. xi, 1861, p. 302. His last paper was on the influence of such compression in causing syncopal insensibility ('Practitioner,' vol. iv, 1880).

also met with in men. This, and the fact that the pneumogastric and vaso-motor systems are readily influenced by emotion, have probably led to the frequent submergence of these attacks beneath the vague conception of hysteria, a conception which conceals whatever it covers. We must rescue from it whatever we wish to study. It can be put back again afterwards if desired. Before describing some examples of these attacks, their special features may be briefly considered.

The vagal symptoms are chiefly sensations referred to the stomach, the respiratory system, and the heart. We may probably ascribe to the gastric nerves a sensation referred to the epigastrium, generally described as a sense of oppression or of fulness, but often indescribable. It begins suddenly, irrespective of the state of the stomach or of its functions, and often seems to ascend to the chest, very seldom to the throat or head, as does the aura of epilepsy. There is seldom nausea and never vomiting. Even more common, especially as an early symptom, is a sense of respiratory distress, of difficulty in breathing. It is sometimes so intense as to amount to orthopnœa, and to compel the sufferer, if lying, to sit upright, although there is no corresponding sign of impairment of breathing. With this may be combined cardiac symptoms, discomfort, acute pain in some cases, often a sensation of sudden stoppage of the heart, followed by rapid action. With the dyspnœa, or the cardiac sensation, or both, is often associated a sense of impending death, so intense that no recollection of its falsity in preceding attacks prevents the conviction of its present reality. It naturally causes alarm, but apart from any cardiac sensation there is sometimes a sense of vague fear and dread, which is recognised to have no adequate cause.

Although there is no impairment of consciousness, a

slight peculiar mental state is common, and may even be the first symptom. It is generally described as a slowness of mental operations, a difficulty in thinking or in concentrating attention. Trifling as it may seem, it always begins suddenly and strikes the patient as a state quite unlike the normal condition. Sometimes it involves a slowness in speaking, but this seems partly due to the sense of dyspnœa. Another occasional feature is a sense of unreality in what is seen. A sudden sense of physical fatigue is sometimes an initial symptom.

The vaso-motor spasm sometimes attains a high degree. To it the symmetrical coldness is certainly due, for the pulse becomes small at the same time. When general, there is pallor of the face. Shivering is common and may amount to a definite rigor, but this occurs when the coldness is beginning to lessen. With the coldness of the extremities, tingling and numbness in them are often described, and sometimes there is slight tetanoid spasm, which we shall consider later.

A fairly typical case was that of a single woman, aged 28 years, whose cousin was epileptic. She had suffered for nine months from the attacks, which were heralded by occipital headache on waking in the morning. Some time in the forenoon she suddenly found that she could not fix her attention on any subject. After this had lasted an hour or so, a sense of sleepiness came on; then suddenly she felt wide awake with an intense feeling of fear, extreme coldness of the hands, feet, and legs, and inability to move. Presently, a peculiar, indescribable sensation was felt at the epigastrium and back, sometimes with slight nausea. After a few minutes of this, violent beating of the heart set in, accompanied almost at once by a feeling of suffocation. If lying she had to sit up to breathe, though there was no evidence of dyspnœa. Then the attack subsided, as a flush

developed, attended by slight perspiration and a flow of tears (without any conscious emotion). The flush was accompanied by a feeling of warmth, beginning in the head and passing to the arms, but with this there was always a slight, distinct rigor. The teeth chattered and the hands shook. It was greater the more severe the attack, but soon ceased, and she was well. During the attack there was a curious sense of unreality in all she saw, and subsequently objects were blurred and indistinct. Some more severe attacks began with tingling in the hands and arms, face, and trunk; the epigastric sensation developed to definite nausea; and general coldness was extreme, "as if made of stone," and the violent action of the heart was preceded by a feeling that it had stopped and that she was dying.

The case I have next to mention is remarkable in its features, although the true vagal symptoms were subordinate to the disturbance of the vaso-motor system. It is notable also from the fact that the subject of such pronounced attacks was a man, an officer in the army, aged 30 years. Two of his sisters were insane. The seizures were not frequent; they had occurred about once in six months for twelve years, ever since he was 18 years old. Their character was always the same, such as one of which I saw the later part. Earlier in the day he had been in specially good spirits—an antecedent often noted. Quite suddenly a dreamy mental state came on, a reminiscent state, the well-known feeling that whatever was happening had happened before. It was not momentary, as in epilepsy, but continued. With it, or just after its commencement, his hands and feet became cold; his own belief was that the mental state came first. With the coldness his face became increasingly pale and physical prostration set in, speedily reaching such a degree that he was scarcely able to move. If he tried to sit up he fell

back at once. His extremities become icily cold, even to an observer. So great was the prostration that he could only utter one or two words at a time, with an effort (apparently from respiratory difficulty in phonation). His pulse became smaller and smaller, until it was hardly perceptible. There was not a moment's loss of consciousness throughout. His own sensation was that he was dying, gradually passing out of physical existence. This state lasted about half an hour, and then he gradually became aware, simultaneously, that his mental state was improving and that his feet were a little less cold. The amelioration went on, but two or three minutes after its commencement a distinct rigor set in, with shivering and chattering of the teeth. It lasted about two minutes and always occurred at this stage, coincident with gradually increasing warmth. A few minutes after the rigor an urgent need for micturition was felt and went on during the rest of the day, a large quantity of limpid urine being passed. No sense of undue warmth or rise of temperature followed the attack; indeed, he continued pale for the rest of the day. Treatment (nitro-glycerine) arrested the attacks entirely, and seventeen years afterwards, I ascertained that his recovery had been enduring. Although he had been in India and had suffered from malaria before the age of 30 years, this could not have been the cause of the rigor, because it was a feature of the attacks from the beginning, before he entered the army.

It is instructive to compare these two cases. Although isolated vagal symptoms were not conspicuous in the second, both present certain features in common—an initial slight mental change, the sense of impending death, the coldness, and the shivering as this was beginning to lessen. While it may be thought possible that in some vagal cases I shall mention, the feeling of coldness may have been a sensation only, independent of the vaso-motor system, in this case

it cannot be thus explained. The vaso-motor derangement was obtrusive, and we shall see that in other instances it was equally clear. The general character of the condition suggests that contraction of the vessels of the brain caused the physical enfeeblement, although the vaso-motor nerves are believed to have little influence on the cerebral vessels. The difficulty in speaking, if of respiratory origin, is another point in common with other cases. When these have been described and considered, I think it will not be doubted that this case is closely allied to the others.

The sense of impending death, so frequent in severe forms, is a mysterious symptom. It has chiefly received attention in connection with the heart, but it seems related also to disturbance of the respiratory functions of the vagus, sometimes to both. In its cardiac associations it naturally arouses a suspicion of angina pectoris, as already mentioned. Anginal attacks were diagnosed in a man, aged 39 years, who had suffered, since the age of thirty-three, from occasional momentary giddiness, even when sitting still, suggestive of the slightest form of minor epilepsy. Six months before he was seen, these ceased and were replaced by peculiar and more prolonged attacks. First he felt a sensation of slight pricking in his hands and feet, combined with a cramp-like feeling; they became colder and colder, and his face grew pale. After about a minute he felt at the heart a peculiar sensation, which he could not describe, accompanied by intense fear and a feeling that he could not get his breath. So intense was the sense of distress that he "hardly knew where he was," but he was certain that he never lost consciousness. After about five minutes in all, the attack gradually passed away. The fact that no pain attended the sense of thoracic distress is a sufficient distinction from true angina pectoris, for which it had been mistaken. The case was doubtless one of the vaso-motor angina of Nothnagel and Powell.

The two cases that follow present the same vagal character, combined with cardiac and vaso-motor symptoms, but without respiratory distress. They differ from the pseud-anginal attacks in the absence of cardiac pain, or any sense of impending death—a difference, perhaps, of degree. They exemplify, moreover, a relation of these attacks to preceding migraine which is not uncommon. A married woman, aged 47 years, with gouty heredity, had suffered from characteristic migraine as long as she could remember. For two years she had been liable to peculiar attacks at intervals of a few days or longer, sometimes as much as two months. A distressing sensation at the epigastrium, described as "fulness" or "oppression," came on quite suddenly and ascended to the middle of the chest; it was attended by an extreme sense of prostration, but there was no pain or nausea. Her heart became excited and her pulse, usually 90, rose to 120; the face became pale, and the extremities cold and clammy; the hands were so insensitive that she could hardly feel what she touched. There was no impairment of consciousness and after ten minutes the attack gradually passed off.

In another woman, aged 52 years, also subject for long to migrainoid neuralgia, attacks occurred which began with a sense of dyspnœa, followed by coldness, especially of the feet, and a sense of "numbness" which passed up the legs and body and involved the arms. The hands became dusky and fixed, with the fingers flexed. Sometimes she was unable to speak for a few minutes. The attack lasted about half an hour and ended gradually.

Tetanoid Spasm.—A slight degree of tonic spasm in the extremities is often associated with vasal spasm, especially when this is so pronounced as to cause tingling and numbness. The spasm is like that of tetany except that

the posture of the fingers is not that of typical tetany, the fingers being flexed at all joints, but this is sometimes met with in otherwise characteristic cases. This spastic feature is conspicuous in some vagal attacks apart from vaso-motor spasm. The influence of even a slight tendency to it is sometimes very obtrusive. It seems to cause discomfort when there is no actual spasm.

This was very evident in one case in which it was marked, and had given rise to much doubt regarding its possible epileptic nature. A lady, aged 25 years, had married at nineteen years of age, and had borne five children during the next four years. The attacks began after phlebitis subsequent to her last confinement, at the age of twenty-three years, and had continued since; sometimes two or three occurred each week. Each began by a sense of difficulty in breathing and coldness in the hands, with some tendency to contraction in them. In a few minutes pain was felt at the heart, the sense of dyspnœa increased, and there was a slight flush on the face. From the onset objects seemed far away, and sounds faint and distant; she was also conscious of an inability to think and a difficulty in finding words: "it was a bother to speak." In one which I saw, she uttered only two or three words at a time, as if she could not get enough breath for speaking, and her aspect was distressed. In this there was no evident contraction of the hands, but she was constantly trying to bend back the fingers of one or the other hand, as if from a feeling of cramp in them. Severe attacks began with deep breathing, and sometimes the sense of dyspnœa compelled her to sit up, with deep flushing of the face, but no duskiness. The hands became very cold, with strong flexion of the fingers. The duration was from five minutes to a quarter of an hour. She never lost consciousness, even for a moment.

The features of this case are typical, and the conscious

difficulty in thinking, as an early symptom, is of interest in connection with the similar symptom in others. The symptoms were peculiar in the dominant tetanoid contracture, which was out of proportion to the vaso-motor spasm. I learned later that the attacks gradually lessened until she became almost free, but were subsequently renewed by an alarming carriage accident.

An instructive connection between ordinary paroxysmal tetany and the vagal form, was presented by a married woman, aged 31 years. A year before she had a long attack of gastric catarrh, with vomiting, in the course of which she had severe tetany, lasting six days, accompanied by elevation of temperature to 102° and 103° F. and a feeble pulse. The fingers were in flexion. The gastric symptoms passed away, but she had another attack of tetany a month after, much shorter; a third three months later, and a fourth after another four months. The latter two attacks lasted only a few hours. Each began with an unpleasant sensation at the epigastrium, followed in a few minutes by contraction on both sides of the face, with the mouth open; the tonic spasm passed to the arms and hands and down the legs to the feet, which were fixed in extension, the toes pointing down. As this developed, the epigastric sensation passed to the thorax and became intensely distressing, giving her the impression that she was about to die. Remissions occurred during the attack, which, after a few hours, passed off in the opposite order to that of the onset. For some days before an attack she experienced, for a few minutes at a time, slight premonitory symptoms. A brief sensation at the epigastrium, with some nausea, seemed to her to pass into the limbs and to cause slight rigidity in them. These symptoms occurred without relation to food or external circumstances. In the severe

attacks the passage of the sensation to the thorax was not attended by dyspnœa; the sense of impending death was not apparently the result of spasm, but resembled that experienced in severe vagal attacks.

It may be noted that in this case no vaso-motor disturbance seemed to be associated with the spasm, and there was no evidence of such a condition elsewhere. The connection of tetany with gastric derangement is frequently observed; in this case it seemed related to disturbance of the gastric nerves only.

Relations to Epilepsy.—The cases I have described present no definite relation to epilepsy. The symptoms were deliberate, slow in evolution, unlike the sudden character of the elements of the epileptic attack. But we meet with cases that seem to connect the two classes, and to establish the position of even the vagal attacks in the borderland of epilepsy. The features of such cases must be individually studied, for they do not admit, at present, of clear generalisation.

A very remarkable instance of combined symptoms of vagal and epileptic character was afforded by a patient who, when a girl of 13 years, began to have attacks at intervals of a day or a week. There was a family history of insanity and probably also of minor epilepsy. Each attack began with a sudden feeling of intense fatigue and with giddiness—a sensation that objects were moving swiftly to the left and changing in colour to purple and red. The feeling made her inclined to fall, but in a few seconds it was over. The colours, the initial sense of fatigue, and brevity distinguished the seizures from simple vertigo. She had also occasional severe headaches, without sensory prodroma. These attacks went on until the age of sixteen years. She was first seen then, but not again until she was twenty-eight

years old. She had married in the meantime and the attacks, which had ceased after she was seen, subsequently recurred in a somewhat altered form, which is peculiarly instructive. Each began as before by a sudden sense of fatigue, which was not followed by the sense of movement and of colours, but by a second's loss of consciousness; sometimes there was only loss of sight, a moment's sudden absolute blackness. These made more emphatic the epileptic nature of the attacks. After another year their character had again changed. The sudden sense of fatigue was followed by yawning, several times repeated, and then loss of consciousness for a second or two, after which she was well. But she did not always thus lose consciousness; sometimes the yawning was followed by a sense of want of breath amounting to distressing suffocation and also by a sensation of peculiar distress below the heart. The effect of these was to make her feel that she must keep perfectly still or she would faint. If she kept quiet the sense of suffocation continued for an hour, or even for two hours, before subsiding, but at any time during its course it might be suddenly ended by a moment's loss of consciousness. The attacks came on when awake or asleep, but if sleeping they were always of the early brief form. She woke up with the sense of fatigue, at once lost consciousness for a moment, and was better. By day they often occurred when sitting still, but sometimes they were excited by rising suddenly from the sitting posture. The brief attacks were clearly epileptic. In those of long duration a vagal symptom was interposed, the respiratory and cardiac distress endured for some time, but was at any time terminated by a moment's loss of consciousness; this seemed at once to relieve the state of brain tension that caused the distress. We can perceive the mysterious relation, although we cannot explain it. The important fact for our present subject is the interposition of prolonged vagal symptoms

in the course of an epileptic attack, as if symptoms, usually momentary, as the aura of epilepsy, become protracted, extended, as it were, into a vagal attack.

In the last case vaso-motor disturbance seemed to play no part. It was conspicuous in another patient in whom loss of consciousness was interposed between the vagal and the vasal symptoms. A married woman, aged 35 years, without neurotic heredity, had suffered for two years. The attacks occurred at intervals of ten days to two months, always without excitant, often when sitting still. A sudden intense sense of suffocation, with a feeling of tightness in the head, was followed, after a minute or so, by a moment's complete loss of consciousness. On its return she felt cold, first and most in the extremities. The coldness was so intense that it made her shiver. After about ten minutes she burst into perspiration; simultaneously her heart began to beat violently and the symptoms slowly subsided. Most attacks were similar, but she occasionally had one that was much slighter and consisted only in a brief sense of suffocation.

An initial sense of fatigue is seldom described in ordinary epilepsy, but we have seen that it occurs in vagal attacks and also in such as we must regard as epileptic and which yet present vagal features. This was marked in the case of a woman, aged 28 years, who had suffered from attacks for three years. At first they occurred at intervals of two or three months, then more often, until of late she had two a week. A sudden sense of great fatigue, succeeded by coldness, was followed in a minute or two by dimness of sight and then loss of consciousness, so sudden as to cause a fall, always backwards. There was no convulsion. After five minutes consciousness began gradually to return. First she could hear loud sounds, then voices, at first indistinctly. Then objects could be seen as in a fog, afterwards clearly. The process of slow recovery occupied

about half an hour. Such attacks bore some resemblance to syncope, but they occurred irrespective of exciting influences. Moreover, she had also slighter attacks with no loss of consciousness, lasting half an hour and closely resembling those of "vagal" character. There was a sudden sense of dyspnœa, respiratory distress, and of faintness, accompanied by a sensation of coldness, especially in the face, on which "icy air seemed to be blowing." Sight became rather dim, and when it slowly returned objects seemed unreal, "as if in a picture"—another feature of vagal attacks.

This sense of unreality, mentioned in the last case, has been already referred to as a frequent feature of vagal seizures. The term was always spontaneously used by the sufferer, and always in connection with that which was seen. It is apparently not the same as the sense of strangeness, the impression that objects and places are unfamilar which should be well known. This is never described in connection with vagal attacks. In these, what is seen is recognised, but seems as if in a picture, as one patient described it. In the last case it occurred as sight was becoming normal after dimness, a fact which suggests that it may depend on some ocular state. For a picture, the focus of the eye is unvaried; it is little varied for a landscape, but with near objects the adjustment is perceptible, and its absence might conceivably cause the impression.

Another illustration of seizures intermediate between those under consideration and epileptic attacks deserves mention. In a woman, aged 38, attacks had occurred which began with an unusual discharge of the special sense centres. A sudden sound, described as a "crack," and referred to the head, was immediately followed by a bright flash of light and a curious brain state, a consciousness of "complete blankness." This continued until she

was roused by shaking. For two or three days after she was more or less prostrate. They were arrested (bromide and nitro-glycerine combined) but recurred, on omitting the medicine, six months later, in the same form but with an additional sensation, that of impending death, so common in vagal seizures. They were again arrested. She had also curious attacks in sleep. She woke to perfect consciousness, hearing every sound and seeing what was visible, but absolutely without the power of voluntary movement, able neither to speak nor move. She was, in truth, one half awake. A vigorous shake completed the process and she could move and speak as usual. But the next day she was always depressed and prostrate.

Extended Epilepsy.—Some of the facts we have glanced at suggest that it is possible that the elements of an epileptic attack may sometimes be extended, drawn out, lengthened, as it were, and thereby made less intense, though not less distressing. To obviate misunderstanding it should be stated that the conception is put forward only as suggestive, as a subject for thought, and the help thought can give to observation.

It is, indeed, difficult otherwise to understand the case described at p. 29, in which prolonged respiratory distress was instantly terminated by a moment's loss of consciousness. If it was a minor epileptic attack it must have been extended, so that the initial sensory stage, instead of being momentary, continued for a long time, and might even subside without the usual loss of consciousness. It is foreign to our thoughts to conceive the idiopathic epileptic attack as capable of extension, and yet we know that the aura varies in duration. In local attacks from organic disease we meet with some that are prolonged, but these are, perhaps, not strictly comparable.

In vagal attacks the symptoms, respiratory and cardiac, which are gone as soon as felt in a true epileptic seizure, seem to be most distressing when prolonged. This can readily be conceived. Full perception is not momentary, and with time there seems to be what physiologists term a "summation of sensation," even of that which is purely subjective.

These facts suggest that if we can conceive a minor epileptic attack that is extended, its elements protracted with no tendency to be terminated by loss of consciousness, its features would be so different that its nature would not be suspected. Velocity altogether alters the effect of momentum. A bullet fired from a rifle makes a round hole in a pane of glass, which it would smash if thrown against it. Swiftness is an essential element of ordinary epilepsy, but this does not preclude the possibility of deliberation.

The attack to be described was, of course, "hysterical." Yet it should be noted that the patient had never had, throughout her life, any hysterical attack of the usual type of such seizures, nor has she manifested any of the ordinary hysterical symptoms. She had suffered from attacks bearing a close resemblance to those of epilepsy. In whatever category the seizure here described is placed, its features deserve consideration not least because an intense conviction of impending death was associated with no special distress to explain it.

The patient was a married woman, aged 35 years, with a nervous system unstable and excitable in a high degree, such as to suggest any kind of hysterical disturbance. She was apprehensive of evil to an extent that made her life distressing to herself and perplexing to others. In childhood she had some attacks that seemed to be definite *petit mal*, with loss of consciousness so sudden as to cause her to fall in each. They ceased after childhood, but there

were subsequently occasional suggestive attacks. One, at thirty-three, began with a sudden sensation of intense coldness at the vertex; she instantly fell unconscious, but at once recovered. The special attack with which we are concerned occurred two years later, after some mental anxiety. She was feeling well, and was occupied as usual, when she had a sudden strange sensation that her mind was acting slowly; an effort was needed to understand what was said to her. This difficulty increased and extended, so that in a quarter of an hour all about her seemed strange and unfamiliar, and an observant nurse who was present averred that for a short time she clearly did not know where she was. She walked into the garden but became worse rather than better. Then, quite suddenly, some intense physical sensation occurred which gave her the impression that she was dying. She begged that her husband should be called in order that she might say farewell to him, although he was then far away. This seemed to mark the turning point, for in a few minutes the physical distress lessened and she began to improve, but for an hour more, when spoken to, she seemed to hear the words at once, but could only grasp their meaning slowly and with considerable difficulty. At the end of one and a half hours from the onset she was quite well again. Throughout the nurse closely observed her and could see no pallor, while her pulse was unchanged in frequency or strength. It was afterwards ascertained that, besides the indications of minor epilepsy already mentioned, she had experienced, many times during her life, and two or three times during the previous year, a sudden sense of strangeness. For a moment she did not know where she was. Once she seized her husband's arm and exclaimed, "Oh, where am I?" but almost as soon as the words were uttered the feeling was gone. This is a familiar form of minor epilepsy. A significant fact is that

she recognised the identity of this momentary sensation with that which developed gradually in the prolonged attack. This, as I said, may easily be regarded as hysterical, but its features suggest that its nature was rather that of a prolonged, slow, cerebral process, which, if condensed into a few seconds, might have involved loss of consciousness and have been a characteristic attack of minor epilepsy. It was attended by intense distress to the patient, and left her with constant dread of a recurrence, but this had no effect in producing one. If its features are compared with those previously described it will be found to present two characters in common with them. It began with a strange sense of a difficulty in mental processes, which is, as we have seen, common in vagal attacks. It also involved the sensation of impending death, which is so frequent in these attacks we have considered, whatever may be the mechanism by which it is produced.

To dismiss it as an hysterical fancy is simply to push it out of sight with the foot. So definite a feeling must have a definite origin. This case shows that the feeling in an intense form may be independent of the sensory disturbance with which it is usually associated, and which is usually regarded as its cause.

The mystery of the cases considered in this chapter seems to increase the more they are studied. They need careful observation in the various aspects they present, and opportunities for this are unfortunately rare. Especially is it necessary that subjective symptoms should receive due weight, and not be dismissed as unimportant.

Treatment.—The attacks without conspicuous vasomotor disturbance are, as a rule, more difficult to treat

effectively than those in which this is conspicuous. The sufferers from these attacks have generally a feeble nervous system, are readily tired, and often prone to over-tax their strength. A quiet, tranquil life is of absolute importance, without fatigue, worry or hurry. Any defect in the general health should be rectified. Anæmia is not often present. Dyspepsia is common and diet should be regulated. The appetite is often bad, and then more food can be taken if it is finely divided and easily swallowed. In some cases too much food is taken; gastric sensations, that are really due to dyspepsia, such as a sense of emptiness and sinking after meals, are misinterpreted, and still more food is put into an overloaded stomach. Flatulence should be lessened by digestive aids, and, if necessary, by hindrance to fermentation, such as is afforded by the sulpho-carbolate of soda. Constipation is very common and especially harmful in these cases; patients are afraid of taking "much medicine," and do not realise that the "much" that is unwise is not the frequency, but the larger dose that is needed if even a day passes without an action, which leaves the bowels exhausted and less inclined to action. A daily laxative, such as extracts of aloes and of cascara, just enough to reproduce normal conditions, induces more and not less tendency to spontaneous action. Others unwisely trust only to an injection, glycerine or other, which does nothing to increase the intestinal secretions to a normal degree, although it is most useful for determining the convenient time for the action of a laxative. In those of gouty inheritance an occasional saline is useful, which "flushes out," and carries away bile that would be re-absorbed. In vagal attacks these measures are very important, commonplace in nature though they may be.

All influences that tend to soothe the nervous system have a beneficial influence on these attacks, and in their treatment we can afford to neglect nothing that is

useful. A course of rest, partial or complete, is often valuable, but isolation is seldom needed. It should be combined, if possible, with fresh air, and with massage, moderate and gentle. That which does most good is the upward movement of the hands, with gentle pressure, and this can be given by any person who has been shown the way, and has the needful endurance of energy. It is purely soothing (the "loomi-loomi" of the South Sea Islanders), while the kneading and punching of skilled massage often fatigues and sometimes irritates the nervous system. Moreover, the upward movement of the hands promotes the circulation in the blood-vessels and lymphatics and the renewal of the plasma that is outside both. Pine-baths have been recommended as having a peculiar tranquillising influence (Hingston-Fox).

The treatment of the tendency to the attacks must be that suggested by the individual condition. In the cases that present an epileptic character, when loss of consciousness forms part of the attack, even occasionally, bromide is generally necessary, and sometimes definitely cures the patient. But these cases are rare, and in the majority, bromide seems to exert little direct influence, although indirectly, for its general tranquillising influence, small doses do good. In the much larger class of cases in which vaso-motor spasm forms part of the attack, and especially when it leads the symptoms, nitro-glycerine is generally of service, often great. This is not by its immediate influence on the attacks (on which nitrite of amyl is much more effective), but by the effect of its regular administration. The dose need not be large; two thirds of a drop of liquor trinitrini, the 1 per cent. alcoholic solution, may be given twice or three times a day, increased if necessary to three quarters or one minim. It should be continued for months, if it does good, and the liq. trinitrini permits its combination with

other agents, provided the mixture is kept acid in reaction. For the first few days the patient is usually conscious of having taken it, even after food, by slight headache for half an hour. Afterwards it is not noticed, even if the mixture is fresh. Occasionally the initial discomfort is greater and the dose has to be reduced, but may be increased in a few days. Dissolved in ol. theobromi, trinitrine may be made into a pill. The liq. is also convenient for giving in a pill, mixed well with pulv. acac. and other agents, any excess of alcohol being allowed to evaporate until the right consistence is attained. Some pharmacists are afraid to triturate the two in a mortar, but, as a fact, the mixture cannot be made to explode by percussion even when dry. The mode in which trinitrine does good in such cases is uncertain. Possibly its regular administration induces a more stable state of the vaso-motor centre. The case described on p. 22 was a striking illustration of its service. In the words of the patient "it acted like a charm." For the same purpose nitrite of sodium may be given, but it has seemed less satisfactory.

Other nervine tonics are often useful, quinine, phosphorus, arsenic, and especially strychnine. Valerianate of zinc, also, has a far wider range of tonic service than its association with hysteria would suggest. In general, it is only by such measures, and a wise ordering of life, long continued, that the tendency to these vagal attacks can be gradually lessened.

The treatment of the attack itself is a subject on which more experience is needed. For the most part, the nature of these derangements has been misconceived by practitioners, on whom we have chiefly to depend for observation, but to whom opportunities do not often occur. If practicable, a warm bath should be given at the outset. When there is chilliness in the extremities, warmth should be applied to them, especially to the legs

and feet or to the epigastrium. If distinct pallor and pronounced coldness, with a small pulse, show definite vaso-motor spasm, an inhalation of nitrite of amyl should be tried, and may be wise even without these indications. Should the subjective distress be great, with the sense of impending death, a few whiffs of chloroform may be given. Chloroform externally, sprinkled on lint, with oil-silk over it to prevent evaporation, applied to the pre-cordial, sternal, or epigastric region, is the most speedy way of stimulating the surface nerves, and will often relieve the cardiac distress, the sense of respiratory difficulty, or the gastric discomfort, respectively. The subjective dyspnœa of breathing may be lessened by inhalations of oxygen, but this measure is seldom available. A diffusible stimulant, such as sulphuric ether, may lessen the symptom in an early stage. Nitro-glycerine has been recommended for relief of the attacks which have an anginal aspect, but its influence is slower and less energetic than that of nitrite of amyl. The momentum that speed confers on energy is needed to neutralise such disturbance as that which constitutes vagal and vasal attacks.

General acquaintance with these affections must become far greater than it is, before effective therapeutic experience can slowly grow and gather weight.

CHAPTER III.

VERTIGO.

VERTIGO occupies a large province in the borderland of epilepsy, perhaps the largest, and that which is the most prolific in instructive facts. The sensation called vertigo is the most frequent subjective evidence of an epileptic seizure, and it is also common from other causes. Its features are similar whatever the cause, and in all forms the sensation may be attended by corresponding motion; there may not only be a feeling of turning or unsteadiness; there may be actual rotation, partial or complete, or there may be a sudden fall, sometimes violent.

The only English word at all co-relative with vertigo is "giddiness"; but this had originally a very different meaning. Vertigo has the definite meaning of "turning," and hence, by a natural extension, it is applied to any feeling or fact of unsteadiness. But the original meaning of giddiness was light-heartedness; whether the easy transition to "light-headedness" has led to the inclusion of true vertigo among its meanings is uncertain. The sense in which it is popularly used is very wide, and includes every peculiar vague brain-sensation, especially brief obscuration of consciousness, imperfect perception of surroundings, and the like. Hence it is always important to ascertain the precise meaning a patient attaches to the word.

As a fact of observation, only one kind of vertigo fre-

quently bears a close resemblance to the epileptic form, and involves definite difficulty in diagnosis; it is that which is produced by labyrinthine disease—aural vertigo. This is also by far the most common, although its nature is often masked by other associations.

The resemblance to epilepsy is, of course, only to the minor form. Whenever there is tonic rigidity or clonic convulsion, the epileptic nature of the case is clear. Although a slight tendency to clonic spasm has been met with when pure aural vertigo was peculiarly intense, the nature of such exceptional cases is otherwise certain. Hence definite convulsion may be regarded as conclusive evidence that any doubtful case is epileptic, and it is seldom that doubt arises in such forms.

The terms "aural" and "labyrinthine" vertigo are practically synonymous. Whatever part of the ear may be primarily affected, it is through the disturbance of the semi-circular canals of the labyrinth that the symptom is produced. The condition is sometimes called "Ménière's disease," after the French physician who first drew attention to the effect of disease of these structures, by describing cases of intense severity, one at least of which was due to hæmorrhage into the canals. It would be wise to restrict the term to such cases instead of applying it, as is sometimes done, to every degree of aural vertigo.

It is hardly necessary to say that the function of the canals seems purely to afford guidance as to the position and movements of the head. The nerves begin in connection with the delicate hairs in the dilated "ampulla" at one end of each canal, which are sensitive to every variation in pressure of the liquid within. Nerve-connected hairs are familiar in the animal kingdom as delicate sensory organs. In insects they seem to receive the vibrations of sound, but in higher animals, although the canals are continuous with the structures by which

sound-vibrations excite nerve impulses, there is no evidence that the canal nerves have to do with hearing. Nor indeed do the impulses excited in them seem to give rise to any true sensation. They seem to pass to a centre where they meet with other impulses from the eyes, muscles, and other sources, with which they agree in normal condition, in a harmony that is unfelt. Any discord between the impressions affects consciousness as a distressing sensation, which may give no suggestion of its origin. Vertigo is the result.

It is, however, a very complex result. The disturbance of equilibrium must be perceived through the cerebral hemispheres, and we can thus understand at least the fact that it is often similar to the vertigo that seems to originate in the cortex—that of epilepsy. In each there may be a sense of movement towards one side, forwards, or backwards (subjective vertigo), or a sense of similar movement in external objects (objective vertigo), or there may be a movement in the sufferer of the same kind, an actual turning or a fall, apparently due to a motor process and not only to the loss of balance (motor or actual vertigo). As a rule all three—objective, subjective, and actual vertigo—are in the same direction. This can be understood on the explanation suggested by Hughlings Jackson that vertigo is a sensation of motor origin. It is a feeling, but due to the perception of a process in the motor centres, or to the results of this. It causes a perception of movement when it is too slight to cause actual movement. In epilepsy we may ascribe it to an inequality in the commencing motor discharge in the two hemispheres, or to the early discharge beginning in one hemisphere only, before consciousness is lost. Such inequality is often manifested in the convulsion by the deviation of the head and eyes towards the side most convulsed.

A slight discharge, too slight to cause any movement,

may yet produce a subjective sense of movement. If it is too trifling to be realised, it may yet produce a sense that objects which are seen are moving in that direction. This will be understood by considering that if a person is actually turning, and an object remains opposite him, he necessarily infers that it is moving also, and in the direction in which he is turning. Thus, even the objective vertigo, the sense of movement in what is seen, is intelligible as the result of a motor process, but is not easy otherwise to understand.

Is the same explanation applicable to the vertigo of labyrinthine origin? On first consideration this may seem different in nature. It has the character of a pure sensation of the disturbance of equilibrium. But so has the slight vertigo of epilepsy, which we can only understand on the hypothesis of motor origin. If we consider, we may see that a motor origin is possible even in labyrinthine vertigo. The nearest physiological analogy is that produced when rotation of the body is suddenly stopped. The tendency to turn continues in the same direction, and may cause a fall. Objects before the eyes seem moving, also in the same direction as the preceding rotation. Yet during the turning, the apparent movement of objects was in the opposite direction. The rotation thus leaves a motor tendency in the same direction as the previous motion, which involves seen objects, as already explained. Apparently the excitation of the semicircular canals by the motion, induces a motor process of the same character in the cortex of the brain. It seems to produce that which would cause it. It is not easy to understand, but we have to accept much that we cannot yet explain.

If the vertigo caused by labyrinthine disease is of the same motor nature, we can perceive why the subjective and objective impressions are in the same direction, and also that the sufferer who falls, sometimes describes his

feeling as that of being hurled down with energy. But the sensation which attends the fall may be complex and remarkable in character, as will be presently described.

One other motor feature has been mentioned, of much significance in relation to this question. A skilled medical observer, who was the subject of most severe aural vertigo, occasionally observed that, at the height of his attack, definite clonic spasm occurred in the arm and leg on the right side, towards which objects seemed to be moving. I have not met with any other observation of a similar fact, but it clearly supports the view that in labyrinthine disease and in epilepsy, the vertigo may be due to processes in the cerebral cortex of more or less similar nature.

Yet we do not know that the feeling experienced, and called by the same name, is always the same in character or produced in the same manner. In some cases the sensory effect may be produced more directly by the disturbance of the lower centres, although there is no evidence that the impressions from the canal nerves ever immediately affect consciousness. The features of giddiness vary much, and are sometimes rendered complex and peculiar by that which appears to be central disturbance, the effect of the derangement of the equilibrial mechanism, and yet affecting consciousness at the same moment. Examples of these associations will be mentioned later.

One fact is difficult to explain, and itself suggests that the process varies: the direction of the subjective and objective vertigo may be opposed. Objects seem to move in a direction opposite to that in which the sufferer seems to turn, or really falls. It is as if the effort to oppose the tendency had more effect on subjective consciousness than the tendency itself; as if a process causing a sense of the movement of objects to the right, excites an effort to correct it so strong as to cause a feeling of motion to the

left. The explanation of these cases may, indeed, be more complex, and to us at present more obscure.

It is necessary to describe the chief facts of the cases that illustrate the several points, because many of these are unfamiliar, and it is desirable to substantiate the nature of the affection. Only the essential facts are given. Significant symptoms are often combined in the same case, in a way that interferes with methodical illustration, and yet has its value, since it impresses on the mind the varied association of symptoms.

In the cases described, hearing was tested up to 2000 vibrations by C tuning forks. By "middle C" is meant that between the treble and the bass, 256 vibrations. The precise number varies with the pitch, and is easier to remember if we call it 250. The C within the treble is C^1, 500 vibrations, and that within the bass C_1, 125 vibrations. Above C^3, 2000 vibrations, hearing is tested by Galton's whistle,* and recorded by the number of vibrations, which are indeed convenient for lower notes. Tinnitus is a symptom of great importance, as evidence of labyrinthine irritation; the word "continuous" is used in opposition to "pulsating," and "constant" is employed in the sense of persistent. The sound of steam escaping from a kettle is continuous, but may or may not be constant.

The features that are common to the attacks of epilepsy and of aural vertigo are these: suddenness, brevity, loss of consciousness, loss of sight. It will be convenient first to consider the occurrence of these in the labyrinthine form.

* By this instrument sounds can be produced which vary from 3000 to 30,000 vibrations per second. It is made by Hawksley, of Oxford Street, and is of great clinical value.

Suddenness.—The actual suddenness with which attacks of labyrinthine vertigo often come on is a fact of importance, because extreme suddenness may suggest epilepsy. One moment the sufferer may be in his normal state, and the next unable to keep upright without support, or on the ground and unable to rise, from the intense giddiness. We cannot ascribe the suddenness of the onset to a sudden change in the labyrinthine impressions. Rarely, indeed, there is an increase of the tinnitus at or before the onset of the vertigo, but this is not common. Usually no change in other aural symptoms suggests a sudden alteration in the nervous influence from the periphery. The suddenness of the onset may be more reasonably regarded as of central origin. It suggests that the equilibrial centre has a certain degree of stability, which suddenly gives way under the cumulative effect of a persistent derangement of the impressions that reach it. We must consider that this centre acts upwards on the cerebral cortex, for it is through this that conscious sensation is produced. Its sudden deranged action may be compared to the process of "discharge" in epilepsy; it is a sudden liberation of nerve energy upwards to the cortex. The constancy of the features of the attacks, in many cases, suggests that, as in epilepsy, the repetition of the process facilitates its occurrence in the same form. Indeed, if the middle lobe of the cerebellum is the equilibrial centre, the facts of many cases suggest the conception of a cerebellar epilepsy, but this conception is mentioned chiefly as an aid to thought; it would carry the analogy farther than is justified. The suddenness of the onset is too familiar to need illustration; it is not so with the feature next to be considered.

Brevity.—We conceive aural vertigo to be of long duration even when sudden in onset, and the vertigo of

epilepsy as brief, almost momentary. But the former may also be brief as well as sudden, and this constitutes one important point of resemblance, which often gives rise to doubt. One difference is that when the severe vertigo is brief, in the aural form, it is followed by slighter giddiness for a time, or by nausea, either of which suggests its nature; and usually the patient has, or has had, attacks which present no resemblance to epilepsy. But it is not always so, as the following case shows.

A man, aged 60 years, had suffered for several years from attacks of momentary giddiness, a sudden impulse to go forwards, instantly over, followed for a few moments by a sense of nausea, equally brief. He never lost consciousness, and had no other form of attack. He had been slightly deaf for six years, hearing on the left side only the highest tuning-fork, 2000 vibrations, and not much higher notes on the right; while for the same time he had tinnitus referred to both ears, at first louder in the left, lately in the right—a continuous hissing, sometimes increased for a moment to a loud "whizz." Such evidence of labyrinthine irritation as tinnitus affords is always important. In this case the aural condition had evidently made little progress during the years of its persistence, and this is of interest in relation to the uniformity of the vertigo. The facts suggest that, as I have just said, a tendency to uniformity had been developed by repetition. Such a case, although not epileptic, is distinctly "borderland." I have seen many similar cases, but must limit myself to those that present specially instructive symptoms.

The sudden brief vertigo often caused an actual fall in a man aged 42 years. The sensation was that everything before him was coming on the top of him. Even when he fell there was no impairment of consciousness; in a few seconds he was able to get up, but a slight sense of unsteadiness remained for ten minutes. Such a fall in

epilepsy would have been attended by momentary unconsciousness. Other attacks, moreover, showed their nature. Occasionally he had time to take hold of something, and did not fall, but he felt himself and objects turning to the right. Such attacks were formerly followed by considerable nausea, by diarrhoea, and also by great coldness. There was clear evidence of labyrinthine changes; hearing had been impaired in the left ear for five years. He had constant left tinnitus, which presented the rather unusual feature of being increased by external sounds. It was "buzzing" in character, occasionally louder, and then attended by a sense of pressure in the ear which seemed to prevent hearing. At the onset of the attacks in which objects seemed to be coming on to him, the noise changed for a moment to a shrill whistle. This affords confirmation of the labyrinthine nature of the attack. Note also the two after-symptoms, both suggestive. One was coldness of the surface, which must be ascribed to superficial vaso-motor spasm. The other is the diarrhœa; it frequently follows the vomiting after vertigo and sometimes mere nausea. It is probably due to dilatation of the intestinal vessels, coincident with the contraction of those of the skin. It is evidence of the readiness with which disturbance of the equilibrial centre deranges the gastro-intestinal tract.

In the last case there was an increase of the tinnitus at the onset of the sudden vertigo, which, as already stated, is rare; in the next the tinnitus was increased, not before, but after the attack. A man, aged 34 years, had moderate deafness and constant pulsating tinnitus on the right side, with continuous slight giddiness. He had also sudden exacerbations of giddiness in which, as in the last case, the floor seemed to rise and pass over his head, and he fell, but without the least confusion of mind. In a few seconds the attack was over, but the tinnitus was much

louder for an hour or more after each attack. Slighter forms, equally brief, were attended by a sensation that he himself was turning to the left. Such a variation in character is not uncommon. The impression that objects are coming on to or over the person is usually a feature of severe attacks, and often causes a fall.

Many of the cases I shall have to mention in connection with other symptoms will afford further illustrations of the brevity of attacks.

Loss of Consciousness.—Momentary giddiness may cause a sudden fall and be mistaken for epilepsy, but the resemblance is much closer when the attack is accompanied by definite brief loss of consciousness. It is not generally known that such loss occurs in purely aural vertigo, and the symptom is often regarded as unequivocal evidence of epilepsy. But the cases to be described afford clear demonstration of its occurrence in vertigo. The fact that this is not generally known, renders it necessary to mention sufficient detail to make the evidence of their nature conclusive. As already said, the condition in any attack of intense vertigo is one of imperfect perception of the surroundings and of their relation to the sufferer, a condition which itself involves imperfect consciousness. We can understand that vertigo, coming on with suddenness and momentum, may for a moment sweep consciousness completely away.

A man, aged 61, had suffered for ten years from bilateral labyrinthine deafness, greater on the right side, with tinnitus louder on the left, and also from attacks of giddiness. They were severe, with a "whirling" sensation. Eight years before, he had consulted Sir Samuel Wilks, who diagnosed "Ménière's disease." The attacks had continued, but had become much briefer, lasting only a

few seconds. Objects and he himself seemed moving to the right, and when these attacks were severe, he fell. Almost immediately afterwards he could recall the giddiness and its character, but not the fall—clear proof of loss of consciousness. On recovery he felt dazed for a few moments, and burst into perspiration. The latter is a common feature of pure vertigo; the "dazed" recovery is a feature of epilepsy. But it is so as an effect of the loss of consciousness, and when this occurs in vertigo a slow return to normal consciousness is not rare.

Another instance of the effect on consciousness of pure aural vertigo is of value on account of the high intelligence of the sufferer. He was a clergyman, aged 37 years, who had suffered for seven years from left-sided deafness and tinnitus, due to chronic changes in the middle ear, spreading to the labyrinth. His first attack of giddiness, three months before I saw him, was intense, making him fall from a chair on which he was sitting, and he afterwards vomited. By a common diagnostic error, the vomiting, which was the pure consequence of the vertigo, was regarded as evidence that the attack was "gastric vertigo." Afterwards he had two slighter attacks, in which objects seemed to go to the left; mere retching followed these. Then occurred one of startling severity. When dressing in the morning before a looking-glass, this and the table on which it was standing, and the floor below, all seemed to rise up and come down upon him, as he fell. He knew of the fall, but was certain that he was then unconscious for a few seconds. He afterwards had others slighter, with objective giddiness to the left, and after each attack the tinnitus was louder, and became pulsating instead of continuous. (This change in character often attends an increase in degree; a uniform hissing, which becomes louder, may beat like machinery.) This patient was a careful observer, and he noticed that an

increase of his tinnitus, with this change in character, often followed, for a short time, the act of rising from the sitting position. This fact is curious. A change of position often induces giddiness in labyrinthine vertigo, presumably because it causes a sudden change of pressure of the endolymph on the hypersensitive structures in the ampullæ of the canals. The pressure of the endolymph must be similarly varied in the part of the labyrinth concerned in hearing, and if the changes are so situated that the influence exerted by rising can act upon them, we may understand that an increase in the tinnitus may be thus induced. Indeed, the pulsatile character of tinnitus must be due to the sensitiveness of the structures to changes of pressure. In this patient the left ear gradually became quite deaf, and then all giddiness ceased.

Again, a man, aged 41 years, had suffered for some years from hissing tinnitus on the left side, on which he could hear no note of Galton's whistle, although it was well heard on the right. Recently he had had attacks in which a momentary "wave" seemed to pass over his head, with a movement of objects to the left; he fell to the left, and was unconscious for a second or two. Sometimes the apparent movement was to the right, and then he fell in that direction. The resemblance of such attacks to minor epilepsy is so close that doubt may be felt whether the association with labyrinthine changes is sufficient ground for regarding the case as essentially vertigo. But before the attacks became so brief he had slighter, more prolonged attacks of giddiness, in which the ground seemed to recede, and if walking he would fall, distinctly from the unsteadiness. These attacks were followed by nausea, and they make the nature of the case certain.

I may mention, lastly, the case of a gouty woman, aged 67 years, who had complete left-sided deafness through

air and bone, with tinnitus "beating like an engine." Attacks of giddiness had occurred during three years, with a subjective sensation of being turned round and round. Lately they had begun very suddenly; they were so intense that for an hour she could not open her eyes, and were followed by prostration and nausea. In one she suddenly fell backwards, but remembered afterwards nothing of the fall; other attacks followed, also with brief unconsciousness, but succeeded by giddiness during many hours, slowly lessening. This patient became much better after a severe attack of gout.

Loss of Sight.—The facts I have described show clearly that intense and sudden vertigo may affect the cerebral functions with such momentum as to cause brief unconsciousness. The sense of a movement of seen objects is distressing in vertigo; so also are visual sensations where there is any subjective sense of movement. The fact that often the patient cannot bear to open the eyes testifies to this effect of visual sensations. It is not, therefore, surprising that when consciousness is not entirely lost sight may be disturbed. In cases of partial syncope, sight may be alone affected, and in epilepsy, sight may be lost before consciousness—and, indeed, may alone be lost. So in some of these epileptoid forms of pure vertigo.

A woman, aged 42 years, with gouty ancestry, had for years deafness of the left ear, distinctly labyrinthine, and also tinnitus—a noise like machinery, which seemed to be behind her. During eighteen months she had suffered sudden brief attacks of giddiness, lasting only a few seconds, in which she felt as if "going heels over head" backwards, and at the moment she could see nothing. There was no unconsciousness. An attack would sometimes occur in bed, and she would then beg to be held

down, so intense was the subjective sense of movement. Although the feeling usually ceased suddenly, a very severe attack was followed by slighter giddiness for some hours, at first sufficient to prevent her raising her head from the pillow. During vertigo, when the sufferer is lying, the act of raising the head usually intensifies the distress. It causes a fresh impression on the nerve endings in the semi-circular canals, which, slight though it must be, is sufficient to intensify the existing disturbance. The fact is easily verified. If a person turns round a few times with the head bent forward he feels sufficiently giddy to have to steady himself by taking hold of some support, but if he raises his head as soon as he stops, the giddiness is so much more intense that he may have great difficulty in preventing a fall.

Sight was lost, for a few seconds, in sudden falls to which a man, aged 61 years, was liable, in which he seemed felled to the ground. They had succeeded slighter attacks of giddiness from which he had suffered for two years. He had middle-ear disease and deafness on the right side, with destruction of the drum, and on the left side he was unable to hear high notes, and he had continuous tinnitus.

The fact that this symptom depends on a special disturbance of the visual centres was shown in a peculiar way by one patient, in whom the loss had the form of transverse hemianopia. A woman, aged 50 years, had suffered for three years from left labyrinthine deafness. There was buzzing tinnitus, pulsating, changing sometimes for a few minutes to a sound of bells. Attacks of vertigo had been severe and distressing, compelling perfect rest for the time. There was a sense of movement to the right in herself and external objects; when lying with her eyes closed, the bed seemed turning over to the right. Later, the attacks became brief, lasting only a few minutes. They began with a "rush to the head" and a sense of

movement within the head so bewildering that she "hardly knew where she was." In one of these, everything was dark in the upper half of the fields of vision for about a minute. Her account was very precise. Such transverse hemianopia is chiefly met with in functional disturbance of the brain, and seems due to symmetrical arrest of action in the region in each hemisphere, related to the upper quadrant of the half fields, and illustrates the close functional relation between the two hemispheres. These brief attacks were seldom followed by vomiting, but generally by great coldness and shivering, no doubt from vaso-motor spasm of the skin, another illustration of the relation of the vaso-motor centre to that for equilibrium.

Sense of Impulsion.—We pass next to a remarkable feature of sudden attacks which is probably characteristic of vertigo, though it may seem to have a close resemblance to an epileptic warning. The very sudden onset, causing a fall, may be attended by a sensation, not of unsteadiness, but that the fall is actually compelled. The intensity involves a sense of compulsion in which the vertiginous element is unrealised. In epilepsy there may be a feeling of involuntary motion, but not of being "hurled to the ground," as patients sometimes describe.

Not only is there often such a feeling of being a passive object on which an external force is acting, but the force may seem to be definite in its direction and even in the part to which it is applied, so as to cause the movement. For example, when gentle, and the patient has a sense of a downward movement, a force seems to act on the top of the head, pressing him downwards. When violent, it is not only more intense, but concentrated and sudden—so sudden, indeed, as to give the impression that a blow has been struck. Except that there is not the super-

ficial pain that a blow would cause, the impression seems precisely to correspond with that of sudden impact.

That considerable vertigo involves a sense of movement independent of the will, and even opposed to it, may readily be verified by the rotation already mentioned. The sensation of continued movement in the same direction is combined with an impression that an external force is at work. This is apparently due to the persistence of the effect of the pressure of the endolymph in the ampullæ of the semi-circular canals. A patient, in whom they had been destroyed on both sides (to cure severe aural vertigo), could be rotated, fast or slow, without the slightest giddiness (see p. 71). The feeling that rotation causes, enables us to understand that spontaneous vertigo may entail an impression of a similar character, of the action of an external force, but it is less easy to comprehend that the impression should have such a degree and character as to amount to the feeling of a blow. It is a remarkable example of what may be called the backward effect of a motor process, generating the close semblance of a sensation, a sensation of an adequate cause of the unwilled effect. There is not time for the development of the ordinary sense of giddiness, so explosive is the onset; its place is taken by the sense of impact. Afterwards the feeling of vertigo is often well marked.

The facts will be better appreciated by the details of some cases. The gentler form of impulsion was presented by a lady, aged 50 years, with slight nerve-deafness on the left side, loud pulsating tinnitus, referred to the head. She had suffered for six months from attacks which began with a sudden distressing sense of downward pressure at the vertex, passing to the front of the head, with a feeling that she was going down, down, through the floor. With this there was a slight tendency to fall to the right, and objects seemed to oscillate from side to side. Thus the

chief subjective feeling of sinking down was accompanied by the perception of a physical force pushing her down.

Much more violent in character was the pseudo-sensation experienced by a woman, aged 34 years, with progressive nerve-deafness and tinnitus on the right side. For five years she had had sudden giddiness, at first simple, and while it lasted she was unable to hear distinctly. Then the attacks began with a sudden sensation of being struck on the head, just behind the vertex. Sometimes she fell, and it seemed to her that the fall was the consequence of the blow. She never lost consciousness, nor had she any definite perception of movement at the onset; the sense of the blow, with the fall, seemed to prevent any vertiginous feeling, but after the fall she was distinctly giddy for an hour, usually with nausea and sometimes with vomiting. The fall naturally caused alarm, and sometimes a sense of dyspnœa. She also had slighter attacks, with the sense of a much more gentle blow and a tendency to fall forward, which also seemed to her the direct effect of the blow. After such an attack there was a slower movement of objects to the left and back again, and she sometimes fell to the left. Occasionally she almost lost consciousness, and once it was actually lost. Afterwards, the attacks occurred also during sleep (a point to be considered presently), and she was once awaked from sleep by a sensation of being struck on the right side of the head, and she seemed to be knocked over to the left by it. She remembered putting out her hands to save herself, but nothing more until she found herself on the ground. Her right-sided deafness steadily increased, hearing being lost from above down until she could only perceive the bass C ($C_1=125$). That the vertigo in this case was purely aural we cannot doubt. The constancy with which the feeling of a blow accompanied the attacks, whether they were slight or severe, is remarkable.

A patient with very pronounced and prolonged aural vertigo once only experienced a sudden sensation of a blow on the ear, making him fall towards the opposite side. The sensation was attended by a momentary pain in the ear that seemed to be struck. Other cephalic sensations at the onset of the attack of giddiness seem to be of the same general nature. One was described as a "clutch," in the case of a single woman, aged 29 years, in whom the aural nature of the vertigo was less certain than in the cases previously described, since there was no deafness. But the patient had persistent tinnitus in the left ear, and such tinnitus means irritation of some of the labyrinthine structures, and this, however trifling, may have a disturbing effect on the centres out of all proportion to its intrinsic degree, and it may be greater in the canals than in the auditory part of the labyrinth. The patient had brief attacks of giddiness, in which the ground seemed heaving up and down, and each began with a sudden momentary sensation of a "clutch" in the head. Later, this clutching sensation occurred when going off to sleep, and was followed by palpitation of the heart; the tinnitus became much louder. Strange to say, her hearing continued perfect, but there must have been definite labyrinthine irritation. The nocturnal sensation, considered alone, bore a resemblance to an attack of minor epilepsy.

Again, a sudden sense of pressure on one side of the head, seeming as if it would throw the patient down, occurred at the onset of attacks of giddiness in a woman, aged 27 years. It was variable, and sometimes not impulsive, lasting some minutes and described as half pressure, half sound, which made her hold her head tightly with her hands. She had the remains of otitis media on the right side and some labyrinthine deafness on the left.

Such cephalic sensations at the onset of an attack of vertigo are often misleading, as they absorb the attention

of the sufferer. Even when the sensation clearly represents vertigo, it may not seem to the patient to do so when it is quite sudden. A lady, aged 60 years, sometimes suffered peculiar attacks soon after rising in the morning. A sudden sensation from the back of the neck seemed to dart forwards over the head, and a feeling that she was losing consciousness made her throw herself on the bed. She never did become unconscious, and in a second or two she was always well. Such attacks, alone, would suggest epilepsy, but we have seen other examples of vertigo of similar brevity, and this patient suffered from loud tinnitus for many months, referred to both ears. She had also, for three months, slight vertigo, lasting several hours at a time, and often severe on first waking in the morning. Sometimes she dared not, at first, raise her head from the pillow; in the night, giddiness was often induced by turning over on the right side, and she was compelled at once to turn back to the left. Her hearing was fair for her age. Thus her symptoms left no doubt that the peculiar brief attacks, doubtful as they seemed when considered alone, were really vertiginous, and so was the sensation darting over the head.

Attacks somewhat similar were experienced, after lying down at night, by a woman, aged 65 years. A sudden sensation of a "rush to the head" caused her instantly to sit up in bed, as she felt she would become unconscious if she continued lying. On sitting up there was at once relief. She had marked aural symptoms, being quite deaf on the right side and partially on the left, and had suffered much from tinnitus, and from slight prolonged giddiness, for six months. These two cases are instructive in the light they throw on sudden attacks with sensations that may readily mislead.

CHAPTER IV.

VERTIGO (*continued*).

Encephalic Vertigo.—A curious feature of some forms of aural vertigo is a feeling of a rotatory movement within the skull. It may be allied to the sudden cephalic sensations just described, and is sometimes equally brief, but often it is prolonged, and so definite that the direction of the apparent rotation can be discerned. It differs from the ordinary sensation of subjective vertigo in seeming to be within the skull, not to involve the head as a whole. The brain seems to the sufferer to be turning over and over, or round and round. In some cases only the posterior part of the brain seems involved. It is not only strange in character, but peculiarly distressing.

Two very brief attacks of this character were experienced by a lady, aged 56 years. Each lasted a few seconds only, and ceased as suddenly as it began. Her feeling was that the brain was whirling round and round from left to right. So intense and startling was the sensation that in the first attack she screamed from fright. Her alarm was the greater since each attack occurred in the night, but a similar sensation, slighter in degree, was occasionally felt in the daytime. As in other cases of the kind, the symptom seemed to be distinctly of aural origin. After the second attack she had constant, slight, vague giddiness, and bilateral tinnitus. There was definite labyrinthine deafness on the left side only.

A similar sensation was frequently experienced by a member of our own profession, a careful and cultivated observer, who has for years been the subject of characteristic labyrinthine vertigo, often so severe as to be incapacitating, with varied and distressing tinnitus. The sensation of encephalic vertigo was confined to the occipital region, a feeling that the contents of the skull in that part were going round in the direction of the hands of a watch. He described it as a "churning" sensation, because it conveyed the idea of rotation against some resistance. (Compare the "grinding" mentioned on p. 62.)

It is difficult to give an explanation of this peculiar form, to conceive why the sense of movement should seem to involve only the contents of the skull. The fact that the structures chiefly disturbed are in the occipital region may be the reason for the limited position of the sensation in the last case. The symptom seems to occur when the labyrinthine process causes irritation in excess of impairment of hearing.

Attacks during Sleep.—Another feature presented in common by epilepsy and aural vertigo is the occurrence of attacks during sleep. It may seem surprising that such disturbance should occur during the cerebral state of sleep, but we have already met with examples, and certain considerations may help us to understand it. That which disturbs sleep is sudden vertigo. The suddenness of the onset, as I have said, we must refer to the centre for equilibration, not to the peripheral processes which derange it. These seem to bring the centre into a state of instability, in which its balance suddenly gives way. During the recumbent posture, usual in sleep, the pressure in the semi-circular canals is different from that caused by

the upright position. If the change in the sensory elements of the canals is such that they are specially disturbed when the posture is horizontal, their stimulation may gradually bring the equilibration centre beyond its stable point. The constant influence at last overturns the balance, as we may often see in the illustration provided for us in flushing cisterns, where a slow flow of water gradually raises a ball to a point at which it turns a tap, and the contents of the cistern suddenly escape. So far we may reasonably speculate. But there may well be more. We know that during sleep some lower centres of the brain are active and less restrained than during the waking state. The impulses from the semi-circular canals will go on, and their effect on the centre for equilibrium will be in relative preponderance in consequence of the cessation of other sensory impressions; the impulses will also vary with every involuntary change in the position of the head, altering the direction of the pressure of the endolymph.

It once chanced to me to have an instructive illustration of the effect of altered pressure. All know, by occasional experience, the strange sleep-sensation of falling—of going down, down some deep space. It often wakes the sleeper, and once woke me up so promptly that I caught the sound of what, I doubt not, was its cause. The sound was the flutter in the ear which is caused by contraction of a tympanic muscle, probably the stapedius. This lessens the pressure in the labyrinth. The ampullæ of the canals being at their lower ends, the pressure in them would be lessened by descent, and its diminution by the stapedius would suggest descent. It would seem, therefore, that even during sleep the equilibrial centre and also the cortex may be influenced by impressions from the canals.

One example of attacks coming on during sleep has

been already mentioned. Another was a lady, aged 33 years, in whom sudden vertigo had occurred for a year, with a sense of motion in herself and objects; if standing she was sure to fall. During sleep she was awakened by a peculiar "grinding" sensation in the head, and a feeling that she was sinking through the bed. She dared not raise her head, and even when the room was dark she had the impression that objects about her were moving. Although she had a brother who was epileptic, her vertigo was certainly aural. Deafness and hissing tinnitus had existed on the right side for eighteen months. Her objective vertigo was of the rather unusual form in which objects seem rotating around an axis in the line of sight, in this case in the direction opposite to the movement of the hands of a clock. It might conceivably be the result of isolated irritation of the right posterior semicircular canal, in which such actual rotation of the head would cause increased pressure.

Subjective auditory sensations have little or no tendency to disturb sleep, if they are uniform. But a sudden loud sound, though purely subjective, will arouse the sleeper, as it would were it of external origin.

The subjects of aural vertigo sometimes suffer from a sudden sound when dropping off to sleep. It is described as a "crash," or as if an explosion had occurred in the head. But it is met with in those who have no apparent aural changes.

Border-line Epilepsy.—We have seen how often vertigo approaches the line that separates it from epilepsy. But epilepsy also may come near the dividing line, at any rate in semblance. True epileptic attacks may begin, not only with the familiar giddiness, but also with tinnitus, the combination of symptoms characteristic of labyrin-

thine vertigo. I have elsewhere * described examples of this, but the following case is specially instructive because there is strong reason to regard it as the result of old stationary damage to the brain, occurring during infancy. The patient was an intelligent girl, aged 18 years, who had a severe brain illness in India at the age of eleven months. This began with a series of severe convulsions; she is said to have been unconscious for a fortnight, and was then unable to move one arm and leg for a few days. It was thought that the convulsions were chiefly on one side, but on which side was not remembered. Her epileptic attacks began at the age of thirteen, and had continued with increasing frequency. Each began with a sudden sense of movement of objects and of herself to the right, and a confused sound in the right ear; with these there was a slight sense of nausea. The warning lasted long enough to allow her to sit down. Then some vague feeling made her close her lips and swallow two or three times. This she remembered, but was not conscious of any peculiar taste or smell. She then lost consciousness, and made a gurgling noise in her throat; her limbs were the seat of slight tonic spasm, sometimes with a few jerks. The whole attack lasted about half a minute, and she was at once quite well.

It is probable that this patient had a small spot of damage to the brain near the auditory centre in the first left temporo-sphenoidal convolution. This centre is excited by a sound which reaches the right ear, and the normal effect is a movement of the head to the right. The instant spread of the discharge to the motor region would cause a sense of movement to the right, and also an impression that objects opposite her were moving to the right. Indeed, the discharge may have involved the left motor region at the same time as the auditory centre.

* 'Epilepsy, etc.,' 2nd edition, p. 234.

Spreading at once to the motor region of the other hemisphere, in the mysterious way the epileptic discharge extends, it caused the slight spasm on both sides, without the deviation of the head and eyes which would have occurred had the motor discharge in the left hemisphere reached a higher degree before it spread to the right. Thus the perception of vertigo in such a case, resembling the aural form, may be regarded as due to activity in the same region of the brain affected in the latter, the motor cortex. The difference is in the nature of the activity; in epilepsy it is a primary discharge; in ear disease it is secondary, a result of the disturbance of the centre for equilibration. We must assume that all sensations of vertigo can only be consciously perceived through the cortex of the brain, whatever be their precise mechanism.

We should note, however, that in the case of epilepsy just decribed, the resemblance to aural vertigo was made more complete by the slight nausea which the giddiness seemed to cause. In vertigo we refer this to the direct influence of the equilibrial centre on that of the vagus, but all lower associations must be repeated in the region of the cortex through which consciousness is influenced. Yet it is remarkable that the sensation should have occurred in association with epileptic vertigo and tinnitus, although it is not met with in the simple vertigo of epilepsy.

Association of Aural Vertigo and Epilepsy.—Few diseases are mutually exclusive. Epilepsy and labyrinthine vertigo must occasionally coincide in the same person without any relation between them. When the epileptic attacks are of the minor form, which vertigo chiefly resembles, the differential diagnosis requires special care. But there may be evidence that the relation is

more than mere coincidence. The attacks, although of different nature, seem sometimes to exert an influence one on the other.

An illustration of the association of vertigo and epilepsy was presented by a member of our own profession, gouty, aged 53 years, whose brother was epileptic. He had suffered for two years from slight nerve deafness and tinnitus. On the left side he could hear no note above 2000. For four months sudden attacks of giddiness had occurred; objects seemed moving to the left, and he fell to the left unless he could support himself, but he never lost consciousness. The intense giddiness lasted one and a half minutes, and for the same time objects seemed to oscillate laterally. He was never sick. For years, every three or four months, he had had a sudden sensation of a pleasant smell, like jasmine, which seemed to fill his nose and head, and lasted thirty seconds, without mental change. Sometimes it was followed by a brief but distressing sense of fulness in the throat and head, so intense that he felt as if "something must give way there." This feeling occurred independently, three or four times a week, by night as well as by day, and at night it was associated with a sense of falling and a sound in the ear. (This seems similar to the stapedius effect mentioned on p. 61.) The sudden brief sense of smell must be regarded as a form of minor epilepsy, and there was some evidence that he once had a definite convulsive seizure.

A more direct relation between aural vertigo and epilepsy seemed to exist in a patient who probably inherited a tendency to the latter, since his father was epileptic. It suggests that, under the predisposition, minor epilepsy may develop from aural vertigo, and in the same manner we have seen reason to believe that it may develop from simple syncopal faints (p. 8). He was a man, aged 38 years, and had become deaf to the watch

on both sides, alike through air and bone. On the left he could hear no tuning-fork; on the right C^3 (2000) was audible only through the air; lower notes also through the bone. For five years there had been tinnitus, bilateral, but greater on the left side, and attacks of giddiness, sudden in onset, lasting an hour or more, attended by vomiting. After two years they became very brief. There was a sudden sense of movement of objects to the left; he fell, and was unconscious for a moment, but the attack was over in two or three seconds. Similar attacks occurred in bed, with a feeling that he was turning to the right, and sometimes there was also momentary loss of consciousness. Subsequently he became liable to attacks resembling minor epilepsy still more closely. They began with what he called a "numb sensation" passing up the spine to the head. If he had time to nip the skin at the back of the neck this would arrest the attack, just as a warning sensation passing up the arm may be stopped by the same expedient. When the sensation reached the head it became "thrilling, like a galvanic battery," and often there was brief loss of consciousness. This case certainly presents the aspect of the development of minor epilepsy from what we may term "epileptoid vertigo," and this from the ordinary form. Such a relation is extremely rare, and the features of any apparent example merit careful consideration. The relation is emphasised in this case by the fact that after commencing bromide he had no more attacks, and his freedom was complete years afterwards.

A remarkable combination of aural vertigo and epilepsy was presented by another patient, whose symptoms were so remarkable as to deserve detailed description. They afford an instructive example of the intricate character such a combination may present. She was a married woman, aged 35 years, who had been slightly deaf all her

life, and had suffered from tinnitus as long as she could remember. It became worse after an attack of scarlet fever at eighteen. At twenty she began to suffer from attacks of giddiness and sickness, each lasting five minutes. At twenty-three she had a fall on the ice, striking her head. At thirty-two she began to have attacks of minor epilepsy, which continued, occurring in the night as well as in the day, and at thirty-three severe nocturnal attacks began. But the tinnitus also went on, and so did brief attacks of giddiness. The minor attacks of epilepsy consisted of lip-smacking and loss of consciousness; at night the sound of the lips always aroused her husband. The severe attacks always began with the same lip-smacking, which was followed by deviation of the head to the right and by spasm—first tonic and then clonic—said to be confined to the right side; after each fit the right arm was weak for a time. There was never tongue-biting, but always micturition. There was no remembered sensation or smell or taste, but a remarkable effect of the severe attacks was complete loss of perception of flavours for two or three days. It was called "loss of taste"; but sweet and bitter were perceived, so that the loss must have been of the olfactory sensation which constitutes "flavour." Yet smell was not lost. I have pointed out* that the warnings of epilepsy indicate a difference in the cerebral representation of olfactory impressions, according to their production through the anterior nares, as a pure smell, or through the posterior nares, as a flavour combined with sensation of taste proper. Although produced through the agency of the same nerve, they are always clearly distinguished by the epileptic patient who experiences one or the other.

Her attacks of giddiness seemed to have no relation to her epileptic seizures, except that there was a sudden

* 'Epilepsy, etc.,' 2nd edition, p. 73.

sense of turning in the same direction, to the right, compelling her to hold on, and of movement of objects in the same direction. With this there was a sound of a shrill whistle going through the head from left to right, "like a train whistling while passing a station"; it gradually died away. These attacks were very brief, but they occasionally caused her to fall. A dazed feeling attended them when they were severe, and once or twice she lost consciousness. They were always followed by brief vomiting. In one she heard the whistle, and felt as if falling down a tremendous height; then she found herself on the floor, having hurt herself in the fall; she was still very giddy and vomiting. The sickness ceased in about two minutes; the giddiness lasted five, and ended suddenly as usual. Sometimes, as the tinnitus was passing away, it changed to a sound of bells ringing. A curious fact is that she sometimes had a peculiar taste in the mouth at the onset of these attacks of vertigo, although never with the epileptic seizure, in spite of the smacking of the lips. The attacks of giddiness had a close resemblance to the paroxysms of aural vertigo, and the fact that they began some years before the epileptic seizures, and continued distinct from the latter, gives some support to the opinion that this was their original nature. But their frequent repetition must have induced a central state, facilitating their recurrence, and it is possible that they had become purely central in nature. The features of the double series of attacks render the case one of unusual interest. The probability that the process had become largely central is important in relation to the next and last class we have to consider.

Pseud-aural Vertigo.—We have seen, in some of the cases considered, how slight may be the definite evidence

of labyrinthine disease, although the symptoms of vertigo are pronounced. In most cases there are tinnitus and impaired hearing to justify the diagnosis of the nature of the giddiness, but in some the only evidence is that furnished by the tinnitus, showing irritation of the nerve endings of the auditory nerve. In the cases now to be mentioned even this is absent as a permanent symptom, although there are paroxysmal attacks of giddiness bearing the closest resemblance to unequivocal aural vertigo. It is, indeed, probable that they are actually of this nature, but the length of time the isolated symptoms existed in some cases, makes it desirable to describe them apart, if only on account of the emphasis thus given to the fact that such cases occur. Although the canals are continuous with the cochlea, they are distinct from it, and they may conceivably be the seat of irritation which the cochlea does not share. It should be remembered also that persistent symptoms of disorder of function do not mean progressive disease.

An illustration of such attacks was afforded by a lady, aged 35 years, with gouty ancestry, who had no tinnitus, except during the attacks, and in whom no impairment of hearing could be discovered on repeated careful examination. Her attacks occurred at varying intervals, a day to a month or more. They were brought on by excitement and over-fatigue, bodily or mental. "Quite suddenly she feels that the ground is rising up to her, and that she is going backwards. This seems to her to be due to some physical force pulling her back. With this this there is a 'rushing' sensation in the head. The feeling of going back lasts for two or three minutes, and its diminution is accompanied by a sound of rushing water in both ears, which ceases when she feels steady. Nausea follows, often for hours, but as a rule she does not vomit. Generally there is no impairment of conscious-

ness," but twice it was lost in an attack—it is said for as long as five minutes. There was never micturition or any evidence of convulsion. A curious feature was that if she resisted the tendency to go back the sense of giddiness was more intense, and afterwards, instead of mere nausea she vomited for a couple of hours. At the early stage of the attack her face was pale and limbs cold; towards its close there was a little flushing. Her attacks were arrested by treatment, but recurred when it was omitted. Its resumption was followed by freedom for five years, when slighter attacks occurred of the same form, but without unconsciousness. Eleven years after being first seen, it was ascertained that she was still quite free from persistent aural symptoms, although slight occasional attacks still occurred. The resemblance of the symptoms to those of unquestionable labyrinthine vertigo is close. The feeling that an external force was dragging her back resembles that of external pressure or a blow, met with in characteristic cases, and only, as far as I have seen, in vertigo of labyrinthine nature. It is difficult to conceive that the attacks had any other origin, but their long persistence in unaltered form is best explained as the result of central co-operation. Central susceptibility may speedily facilitate repetition in varied forms of functional disorder. Such an associated disturbance of function may have been first set up by trifling peripheral derangement, and have gradually become independent of that which produced it, and purely central. The process is comparable, for instance, to convulsions caused by tape-worm, but persisting, after perfect expulsion of the cause, as permanent epilepsy. All peripheral associations are reproduced in higher centres, and hence their association must not receive too much weight as evidence of peripheral disease. But the doubt that may be felt regarding the nature of these cases makes

it wise to keep them apart—at any rate until more is known.

In another case, a man, aged 38 years, central disturbance probably played a considerable part in the attacks. They occurred about once a fortnight. A sudden sensation darted over his head from behind, and was at once followed by slight deafness and hissing tinnitus, while objects before him seemed moving from left to right, without any sense of movement in himself. The duration was thought to be about two minutes. Nausea followed but no vomiting. Sometimes brief tinnitus occurred alone, and sounds for the moment seemed distant. There was no constant tinnitus, and not the least impairment of hearing. The further course of this case is not known.

Treatment.—The general treatment of labyrinthine vertigo is scarcely within the subject now considered, but a brief account of it is necessarily involved in the treatment of the borderland forms.

Local measures can do little for labyrinthine changes. Such as are demanded by primary disease of the middle ear belong to the province of the aural surgeon. The bold resources of modern surgery have, indeed, effected the absolute cure of most intense labyrinthine vertigo, with distressing tinnitus, by the destruction of the semi-circular canals.* The operation was performed on both sides, because there was bilateral disease, and the balance of loss is essential for equilibrium. The result was perfect, and no rotation, slow or quick, gave rise to any sense of giddiness. But the operations involved destruction of the cochlea also, except a small portion on one side, so that it produced permanent deafness almost complete. The choice it involves is thus between two grave disasters, and

* Lake, 'Med. Soc. Trans.,' vol. xxvii, 1905, p. 364.

one element in the decision may justly be the fact that, as in the case referred to, the patient's age permits the effective acquisition of the "lip-language."

If the labyrinthine affection is primary, local measures are almost limited to counter-irritation over the mastoid process, by a series of small blisters, one a week, or more frequent mustard leaves. Some benefit is occasionally obtained, but it seems to have more influence on the tinnitus than on the vertigo. It is always important to consider whether any diathetic state underlies the labyrinthine changes, and to treat it, in the hope of at least staying their further increase, possibly promoting their diminution. It is very seldom, however, that the condition can be ascribed to any general state on which treatment exerts marked influence. If such chronic changes are ever due to syphilis, this cause must be extremely rare. They seldom present the subacute course that would characterise a syphilitic process on which treatment can have much effect.

A gouty state, or gouty heredity, is often to be traced in the chronic cases after middle life, which constitute the majority. We know that this tends to induce changes in the fibrous tissues, such as form the membranous labyrinth. It seems also to promote the early occurrence of the changes in such structures which age induces. Organic alteration we cannot hope to remove, and the tinnitus that attends such conditions, though often lessened by treatment, seldom ceases entirely, except in the very rare cases in which the disease is so progressive as at last to abolish all hearing. Yet in some cases it diminishes, in time, to a remarkable extent, and the distress it causes is so largely influenced by its degree that a slight diminution often makes a disproportionate difference to the comfort of the sufferer. The tendency to giddiness is much more frequently influenced by treatment than is the tinnitus.

In the patients in whom a gouty diathesis is manifest or probable the mode of life should be carefully regulated. The details need not be here described, but saline aperients are of great importance; mere regularity of action of the bowels is insufficient to lessen definitely a gouty state. Whatever the part played in it by uric acid, the deposits of insoluble urate of soda have clear significance, and small as the amount of soda may be in certain aerated waters, if these are taken daily, in time a definite difference must be effected by the substitution of such as contain potash or lithia, which form soluble salts with uric acid, for those containing soda. Lithia may also be given internally, or the new solvent for uric acid, thyminic acid, which seems distinctly useful. The process in the labyrinth is seldom of a character to suggest the use of colchicum, but small doses of mercury may often be given with apparent advantage, and continued for a considerable time.

The study of the symptoms has brought prominently before us the part taken by the central structures in determining the occurrence and character of the attacks of vertigo, and it is on these that most influence can be exerted. We have seen that the suddenness of onset must be referred to the sudden disturbance of the stability of the equilibrial centre, and bromides certainly enable us to lessen the readiness with which it is deranged, to reduce the frequency of the attacks, and also their severity. Several of the cases I have mentioned afforded evidence of this effect, and in some arrest was obtained and continued as long as treatment was continued. We may, at least provisionally, conceive the process as an upward "discharge" from the lower centres, acting on the cerebral cortex, and inducing in it motor and sensory activity, by which consciousness arises of the features of the attack. The conception is indeed largely specula-

tive, but it is the best idea we can form of the process that occurs. More or less suddenly, energetic action occurs in the nerve centres. When such action seems spontaneous we call it "discharge," and the same term is justifiable in this case, because any exciting cause that we can discern is inadequate for the effect. It is therefore reasonable to employ the same agent, bromide, that is so often effective in the unexcited activity of epilepsy, and the result justifies its use, at least in many cases. It may also act beneficially on the secondary process in the cortex itself. To produce a distinct effect it is necessary to give not less than thirty or forty grains of bromide daily, and sometimes more is necessary. In those who are gouty the bromide of lithium may be reasonably preferred, although it is doubtful whether much of the base is released in the system. The beneficial influence seems often to be increased by the addition of some sedative agent, such as belladonna, gelsemium, and especially hyoscine hydrobromate. The last may be given in doses of four or five minims of the 1 in 1000 solution, which is the most convenient mode of prescribing it by the month. When the pulse is rapid, and especially when the pulsatile character of the tinnitus suggests undue sensitiveness to the arterial pulsations, digitalis or strophanthus may be added with advantage.

It is important to avoid all agents, such as bark, quinine and the salicylates, which have an irritant action on the labyrinth. The administration of such to excite disturbance of the labyrinth, in the hope that the subsidence of the disturbance might leave the nerves more quiet, has only to be mentioned as condemned by experience. It was advocated by Charcot, but certainly on insufficient grounds. The same opinion must be expressed regarding treatment by injections of pilocarpine to produce copious perspiration, which was once advocated but soon discontinued.

We do not know whether the sedative treatment, which certainly most frequently does good, exerts any influence on the nerve endings themselves, and the disordered impulses they convey. Bromide, at least, has an effect in proportion to the amount of central co-operation that is suggested by the symptoms. Hence it is always important to endeavour to discern the part the centres play in the disturbance, as indicated especially by uniformity in the character of the attacks. The central influence is probably least, and least benefit can be anticipated, in the cases in which the aural symptoms present evidence of steady increase, and the attacks are variable in the features they present.

CHAPTER V.

MIGRAINE.

SOME surprise may be felt that migraine is given a place in the borderland of epilepsy, but the position is justified by many relations, and among them by the fact that the two maladies are sometimes mistaken, and more often their distinction is difficult.

The term "migraine," or "megrim," originally "hemicrania," is strictly applied to one-sided pain, the typical form of paroxysmal headache. But we cannot, practically, separate this from general headache of periodical occurrence. Both occur with the same associations, have a similar course, and may occur in the same subject. One may pass into the other. An inevitable result of this is that the word "migraine" is losing its original use—that is, its application to strict "hemicrania," and is employed as a name for paroxysmal headache in general. All forms may be preceded by similar sensory symptoms, and may lead to vomiting. The following description of its relations to epilepsy does not pretend to be an account of the malady as a whole.

Alternation.—One definite relation between the two diseases is that they sometimes alternate—one replaces the other in the same subject. If a person subject to migraine becomes liable to epilepsy, the migraine may

cease, or become slight and rare, and return if the fits are arrested. This was the case in a woman, aged 32 years, who at the age of eight became subject to periodical headaches—general, not one-sided; each was preceded by dimness of sight, and usually ended, after some hours, with vomiting. They continued until, at the age of thirty, she became subject to epileptic fits—severe and frequent. The headaches ceased entirely when the epilepsy began, and remained absent until she came under treatment. By this the fits were arrested, and during eighteen months she was under treatment she had attacks only once. But the headaches returned soon after the arrest of the fits, and they were of greater severity. The pain extended from the head down the sides of the face, and lasted the whole day. During these headaches—not before them—she often had brief impairment of sight for a short time, sometimes in one half of the field, sometimes in both, an instance of the fact that the sensory disturbance which usually precedes the headache may also occur during its course.

Another example of alternation was presented by a member of our own profession, who was thirty-eight when first seen. There was a family history of gout and migraine, not of epilepsy or insanity. He had suffered from characteristic migraine, which began at the age of fifteen; the headache was preceded by visual spectra. At the age of seven he had some brief attacks of micropsy, in which objects seemed small and far away, without headache. (This is an occasional aura in epilepsy and may occur alone.) When twenty he had attacks in the early morning, consisting of a peculiar flavour and a brief dreamy sensation for a few seconds. After these had gone on for a few weeks a distinct epileptic convulsion occurred in the afternoon, but no more. Other minor attacks followed, now a sense of smell, not of flavour, and a brief confused feeling, instead of a "dreamy" state. These

ceased and the headaches returned, which had been absent during the epileptic period, but they were no longer preceded by the visual sensation. They ceased, however, on the return of still slighter minor attacks, consisting only of a brief inability to speak. After a time these also ceased, but there was no subsequent recurrence of the headaches.

But epileptic attacks and paroxysmal headaches may co-exist, even when the headaches are preceded by definite sensory symptoms. Instances of such concurrence will be presently mentioned. The alternation is also common, and further examples will occur in connection with other symptoms.

Premonitory Symptoms.—Sensory symptoms often precede the headache of migraine and the attack of epilepsy. The most frequent before headache are visual symptoms, which are less common as the aura of epilepsy, but not rare. The two differ essentially in duration—before epilepsy brief, almost momentary, before migraine deliberate, occupying a quarter of an hour. Epileptic attacks are preceded by a far wider range of visual sensation, from simple colour or simple stars or general loss to elaborate visions of persons or places. The sensory premonition of migraine is either simple loss, varying in area, or luminous discharge of very varied pattern, but never attaining a higher elaboration. One of the most common may be mentioned. A small star appears near, but not at, the fixation point, it enlarges towards one side, its rays expanding into zigzags, often coloured—the "fortification spectrum." Within it vision is dimmed by bright scintillation. It becomes faint when it has almost reached the periphery, and ends in various ways which are not relevant to our present object. It lasts a quarter of an hour or

more, and as it ceases the headache comes on, usually on the opposite side of the head. Sometimes the visual disturbance is central and bilateral. Many other forms are described and illustrated in my Bowman Lecture on "Subjective Visual Sensations."*

These visual symptoms are well known and are sufficiently distinctive. It is not so, however, with other premonitory symptoms which occur in the periphery, especially the arm and the face. A sensation of tingling, often like "pins and needles," is felt in the hand and slowly ascends the arm, leaving behind it "numbness," diminished sensibility. It occupies about a quarter of an hour in passing up the arm, and as it ceases the headache begins on the opposite side of the head. The ascending zone of tingling obviously corresponds to the expanding luminous zigzag in the field of vision, and the impaired sensibility left behind it is similar to the dimness of sight within the zigzag. We can scarcely doubt that the symptoms are due to the same process in different regions of the brain, in the visual centre, and in that for sensation in the arm. A similar sensation may be felt in the face, especially near the mouth and in the lips, either after ascending the arm or independently. It may be felt in the lips on both sides, and with the lips the tongue is often involved. Moreover, when the prodroma occurs in the right arm it may be accompanied by an aphasic difficulty of speech.

The sensation passing up the arm resembles, except in its slowness, the aura of epileptiform fits caused by organic disease of the cortex of the brain, either active disease, as a tumour, or an old stationary lesion in or near the arm centre. Such a sensation precedes the convulsion that begins locally in "Jacksonian convulsions," or in "organic

* 'Trans. Ophth. Soc.,' 1895. Reprinted in 'Subjective Sensations of Sight and Sound, etc.,' London, Churchill.

epilepsy," as the recurring fits from an old stationary lesion may be termed. The resemblance is important for many reasons, practical and pathological. One practical reason is obtrusive; in spite of the difference in duration, the symptoms of migraine may be and have been mistaken for those of organic brain disease. Since the mistake may be made by men of experience, the causes of such error need consideration.

The process which gives rise to the sensory symptoms that precede the headache of migraine is very mysterious, whether it is referred to the eye or the arm; there is a peculiar form of activity which seems to spread, like the ripples in a pond into which a stone is thrown. But the activity is slow, deliberate, occupying twenty minutes or so in passing through the centre affected. In the region through which the active ripple waves have passed, a state is left like molecular disturbance of the structures. There is a subjective sense of "numbness" in the arm and a peculiar scintillation in the field of vision, but the structures affected are less susceptible to stimuli reaching them from without, so that there is dimness of sight in the eyes and defective sensation in the arm. It slowly subsides to the normal condition. Then comes on the pain, often so intense, on the side opposite to the peripheral symptoms. We must refer the latter to disturbance of the cortical centres of the brain, but we know nothing of the precise nature of the process. It has been conjectured to be due to local arterial spasm, but of this we have no proof. Contraction of the peripheral arteries commonly attends the pain as it develops, causing pallor and coldness of the surface, but this is apparently an effect of the pain, as the arteries of a frog's foot contract when its brain is injured. The opinion that it is due to local vaso-motor changes is difficult to reconcile with the character of the "discharge," so uniform in its general character, so

orderly in its disorder. Not less mysterious is the pain, so intense, often so restricted, and felt at the seat of the disturbance which causes the peripheral symptoms, in the side of the head opposite to that to which the sensory derangement is referred. But the pain does not concern us now.

Distinction from Epilepsy.—The important fact is that a sensation beginning in the hand and passing up the arm occurs not only in migraine but as the aura of convulsions due to organic disease of the cortex, although the two differ in their duration. The epileptic aura occupies a few seconds, the premonition of migraine is almost always many minutes, often twenty or more, in its course. Another point of resemblance is that a sense of weakness may accompany the sensory disturbance in each malady. Each may have headache as its sequel, but the pain of severe migraine is incomparably greater than the headache which follows a local epileptic seizure too slight to develop to convulsion. When the right arm is the seat of the sensory disturbance, transient aphasia may be another source of confusion. But the duration of the symptoms affords a decisive indication. In the following case this was so clear that the facts would scarcely be worth description were it not that the nature of the case was actually misconceived. A boy, aged 13 years, belonging to an intensely gouty family, for several years had been subject to periodical headaches, formerly general or vertical, but of late only on the left side. (Here we have an illustration of the fact already mentioned, that we cannot separate the periodical headaches that are bilateral from those which are "hemicranial.") When the pain in the head became limited to one side each attack was preceded by a sensation in the right arm, the characteristic tingling, beginning in the hand, passing up the arm, and

leaving numbness behind it. It occupied a quarter of an hour in its transit, and with it there was distinct aphasic difficulty in speaking. As it ceased, the left-sided headache began; and it lasted some hours. These symptoms were typical; and yet an experienced physician expressed the opinion that the case was one of focal epilepsy, "Jacksonian epilepsy," probably due to a tumour of the brain, and advised an operation. The diagnosis of migraine was entirely confirmed by the subsequent course of the case.

Besides the difference in duration there are other distinctions. The sensory disturbance in migraine may affect both hands, but this is never the case in Jacksonian epilepsy. The aura of this may start in the foot or the hand; that of migraine very seldom begins in the lower limb. I have never met with onset in the foot, and I have once only known the pre-migrainous sensation to pass to the leg. The epileptic aura, ascending the arm, seems to pass to the face or the head, but that of migraine is never thus described. On ascending the arm it may be felt in the lips independently, sometimes simultaneously. As just mentioned, it is often felt in both sides of the lips, and usually in the tongue. This is of interest, because the lips are essentially bilateral in function and are closely related to the tongue.

Although it is rare for headache with premonitory visual symptoms to be thought to be epilepsy, the mistake was made in the case of a man, aged 38 years, who had suffered from general epileptic fits at night only, at intervals of several months, since the age of seventeen. His headaches were preceded by visual symptoms, "haziness over the sight, with bright specks in it," which lasted five minutes or more, even up to an hour. It impaired his vision, so as to prevent him going on with work. Before this ceased the headache began, occipital and slight at first, but becoming intense, and at last

localised to the right eye. The fact that the patient was known to suffer from nocturnal epilepsy, led to the conclusion that the visual symptoms in the day were also minor epileptic attacks. The mistake is a useful illustration of its class. It is reasonable to assume that all paroxysmal symptoms are of the same nature; but this assumption should never prevent their careful scrutiny, which would at once have revealed the nature of the visual sensations, in spite of their difference from the type.

The same mistake of regarding the symptoms of migraine as those of epilepsy was made in another case, but with more excuse. A boy, aged 16 years, had suffered for a year from attacks which were regarded as certainly epileptic. Each began by the appearance of misty blotches in his sight, without colours or spectra of definite form. This amblyopia continued for half an hour; as it passed off pain came on in the left side of the forehead, increasing to great severity. After two to four hours, vomiting occurred, and immediately the pain lessened and soon passed away. So far the symptoms are typical. But a few minutes before the vomiting, after the headache had lasted some hours, a sensation of "pins and needles" was felt across the upper lip and in both hands. In the earlier attacks the sensation was felt only in the first and second fingers, but in the later ones it spread over the whole hand. It accompanied the vomiting and ceased with it. This is another instance of the occurrence of sensory symptoms during the course of the headache, and this unusual feature helped to give rise to the diagnosis of epilepsy. It seems to have coincided with the relief of nerve-tension by the vomiting, which often seems to free the sufferer from the pain. As in the previous case, the sensation in the lip was bilateral. The partial and symmetrical affection of the hands is one of the peculiar features of the sensory disturbance of migrane, and when

bilateral it is distinctive. The case illustrates also the perplexity that may arise from a variation in the order and character of the symptoms. We shall see other examples of this, and their consideration will assist the discernment of the nature of other unfamiliar symptoms of this most variable disease.

Isolated Prodromas.—It is important to remember that the sensory prodroma of migraine may occur alone, without succeeding headache to emphasise its nature. Any form may be thus isolated, and may sometimes give rise to perplexity.

A visual sensation occurred without a succeeding headache in a man whose symptoms presented other features of interest. He began to suffer from migraine at sixteen, and had two epileptic fits during the six months before I saw him at twenty-four. The headaches had always been preceded by hemianopia, often on the left side, lasting ten or twenty minutes, followed by pain in the right eye and right side of the head. Still more frequent, however, was transverse hemianopia, loss of the upper half of the combined fields. (I have mentioned this remarkable feature in connection with vertigo, p. 54.) The loss, whether lateral or transverse, was attended by a "flickering," but never by a *zigzag* spectrum, although occasionally there was a whirling circle, "like a Catherine wheel." The succeeding headache lasted two or three hours or longer, even the rest of the day. Afterwards, he frequently had the visual symptoms with very slight and brief headache, and sometimes with none. They were induced by a sudden change of light, especially by going into bright sunlight; sometimes by going into a darker place. When thus induced, and without headache, they were much briefer, lasting sometimes only for a minute,

and often less than five minutes. In such detached form they might easily have been thought to be minor epileptic seizures, but the identity of character with those that preceded the headaches made their nature certain. He afforded, also, an illustration of the real difficulty which such cases sometimes cause. He had one attack unlike the others. Suddenly, voices seemed far away, his field of vision became lessened on each side, and he was extremely pale, as if fainting, but presently recovered. He had no others like it, and its nature is uncertain. He had only one more epileptic fit, and his headaches became much less frequent during the eighteen months he was under treatment.

The cases in which such isolated sensory disturbance is in the arm, especially in the right arm, and is attended with definite aphasia, may give rise to great perplexity, although not quite relevant to the present subject. The symptoms seem like those of sudden arterial obstruction in the brain, causing transient hindrance to the blood supply. The fear is specially aroused, if there has been no previous similar attack. Subsequent headache, if it occurs, may not lessen the concern. The difficulty is greater if the patient presents conditions favourable to arterial obstruction, if he is in the degenerative period of life, if he has had syphilis, or presents any sign of heart disease. The guiding points are the following. In most cases the sufferer has been liable to paroxysmal headaches, with or without preceding visual spectra. The sensory symptoms in the arm predominate, and their peculiar character, as I have described it, is really distinctive, if only the fact is known. So also is a sensation in the lips, subsequent or simultaneous. Lastly, the symptoms seldom last more than half-an-hour, and at the end of an hour all is normal, unless some headache remains. It requires courage on the part of a doctor, confronted with

such symptoms for the first time, to dismiss them as functional, because he knows that he will be blamed should they be the harbinger of grave disease. In the degenerative period of life, indeed, symptoms of this nature may have the feared significance, but then they are not so transient. A few days since I heard of a lady whom I saw some time ago, who was in late life the subject of migraine with a visual premonition, and who had sudden weakness in the right arm and some aphasia. They did not pass away until a week afterwards, and were evidence of a slight organic lesion. But the arm was weak, not tingling, and the duration of the symptoms was clear proof of their nature.

Vertigo before Migraine.—It is not common to meet with vertigo as a premonitory symptom of headache, and when it occurs its rarity may render it a cause of perplexity. Usually slight, in those who are subject to it the vertigo is occasionally more severe, and its nature may be easily misunderstood. Even when severe enough to involve actual instability, it is seldom so definite in character as to permit a description of its features. The following case is an example of prodromal vertigo, and also of another feature, the variation in duration of the premonitory symptoms in the same patient.

A man, aged 30 years, had been subject to migraine since he was a boy. The headaches were bilateral and preceded by loss of sight, usually general, sometimes hemianopic, which lasted only a few minutes before the headache came on. Sometimes, instead of the affection of sight, there was numbness of the lips or of the tongue or of both, equally brief. These symptoms occasionally occurred without a succeeding headache. With them there was often a slight vague sense of giddiness. On

one occasion, two months before I saw him, there was a much more prolonged warning. It began with such severe giddiness that he could not stand, attended by flushing of the face, with a pricking sensation in it and in the right limbs. After half-an-hour it lessened, but returned in a few minutes with renewed and more severe "pricking" in the right limbs; this seemed to cause weakness in them. The symptoms gradually lessened and passed away, but before the giddiness had ceased the headache began, of the usual character: at first a sense of pressure at the vertex, gradually passing into severe pain, much more intense than he usually experienced, which continued for twelve hours.

Such a long prodroma bears a resemblance to the symptoms of an organic lesion rather than to epilepsy, and this was increased by the weakness that attended the sensation in his limbs. It is important to remember that any sensory discharge may exert an inhibitory influence on the motor centres. In this case the symptoms differed only in degree from the usual premonition, and the headache was precisely that from which the patient had habitually suffered. The brevity of the premonitory symptoms in the ordinary attacks may seem a resemblance to epilepsy, but their character was distinctive. Tingling in the lips or tongue on both sides is confined to migraine, as I have said. Lateral hemianopia is a symptom which, strange to say, seems to be unknown as the aura of an epileptic attack, although a brief transverse loss is occasionally met with. In one case the aura of the fits was a sudden green colour in the lower half of the fields of vision, from side to side; to the patient it seemed as if "he was standing in a field of grass."

Symptoms during Pain.—Another class of symptoms which may sometimes bring migraine near epilepsy, and frequently seem to do so, is disturbance of other brain functions during the pain. As I have said, we do not know by what mechanism the pain is caused. The fact that when unilateral it is on the side of the head opposite that to which the sensory symptoms are referred, must indicate a direct relation to the substance or membranes of the hemisphere. This fact enables us to understand that signs of other disturbance of function of the cortex should sometimes coincide with the pain. They seem to result from its intensity and always coincide with its extreme degree. One such effect is very common—the oversensitiveness to sensory impressions which accompanies all severe attacks. The pain itself is increased by light and sounds, so that darkness and silence are needed.

Somnolence.—One rare effect of the pain is sleep. It is so rare that it may not be right to speak of it as an effect, although it has this semblance; it may be another manifestation of the brain disturbance that causes the pain. When it occurs it occasionally entails peculiar difficulty in the diagnosis. It was so in the very instructive case of a boy, aged 11 years when I saw him, whose symptoms had been confidently ascribed to epilepsy. A year before, at a party, he struck his head against that of another boy. The blow was near the left parietal eminence, but not severe. He had immediately some difficulty in speaking, and headache came on quickly. A few hours afterwards a doctor saw him: he could then speak well, but the pain was severe, and he had begun to vomit. There was no sign of injury to the head. He went to sleep, and next day was quite well. He continued so for six months, when, one morning, he started alone for a walk along a country road; in an

hour, he was found by the side of the road, fast asleep. He was roused with difficulty and taken home; at once he went to sleep again and slept for several hours. He then said that he had had a little headache on starting; it increased, he felt he must lie down, and at once went to sleep. A month later, headache began in the morning, slight at first, gradually becoming severe, and again he went to sleep for several hours; when he woke up, he was violently sick and then was better. Half an hour after the headache came on, he felt a sensation of tingling in his lips and spoke with difficulty; his lips seemed stiff and could not be moved properly. This lasted about an hour, and passed off before he went to sleep. After another attack he was feverish, with a pulse of 120. The headache always began at a spot just behind the left parietal eminence, and spread thence forwards and backwards, but never went to the other side.

That the case was one of migraine admits of no doubt, and was confirmed by the effect of treatment; bromide was useless, trinitrine and strychnine arrested all attacks. It presents many instructive features. The excitation of the first attack by a slight blow on the head is unusual, and was misleading; the immediate symptoms might reasonably be ascribed to damage to the brain. The deep sleep in the second and subsequent attacks bore a suspicious superficial resemblance to the post-epileptic sleep, and vomiting is not uncommon after convulsive attacks. But severe headache never follows attacks too slight to be noticed; while vomiting is not a sequel to such slight seizures, nor does it follow prolonged post-epileptic sleep. It occurs soon after the attack. The lip symptoms occurred after the onset of the headache, instead of before it, as did other symptoms, usually initial, in a case already mentioned, and it is probable that the difficulty in speech was due to an inhibition of the lip movements rather than

to actual contraction. A similar inhibition may attend the sensory symptoms in the hands, as we have already seen. One other feature also deserves note, though it is not connected with our present subject; it is the feverish condition which attended one attack. There is no doubt that in young children, migraine is represented by feverish attacks, with headache, which bear an alarming resemblance to meningitis, but pass off in about twenty-four hours, to recur after a few months. These attacks may be replaced by simple headaches in later childhood. Apart from this, the case shows how easily the features of migraine may mislead. The first attack, indeed, excited by a blow, could scarcely have been interpreted aright.

Delirium.—The intolerance of sensory impressions, which the pain causes, may pass into more pronounced cerebral disturbance. The intense suffering may induce a state of stupor, in which the pain seems to be felt as acutely as ever but is not afterwards remembered. The condition is often attended by quiet delirium of which nothing can subsequently be recalled. The patient is usually said to be "unconscious." It must be remembered that the word "unconsciousness" is used as loosely as "giddiness," and is applied to delirium as often as to stupor. Such delirium was presented by a married woman, aged 30 years, whose mother suffered from headache, and father from gout. "Sick headaches" began when she was a girl, and became more severe at twenty-three, and gradually more frequent. They began in the morning suddenly; the pain was at first occipital and later extended forward over the eyes. No sensory symptoms preceded the attack, but an unusual feature of the pain was that it compelled her to sit up, being greater when she lay down. As it grew most intense she became paler, her hands and feet extremely cold, and she passed

into a delirious state, making strange statements, of which she afterwards remembered nothing. Her condition was described by a doctor who saw her as resembling epileptic mania. As the pain lessened her mental state became normal.

A similar condition, more variable in duration, occurred in another woman, aged 28 years when first seen ; her headaches were then attended by similar delirium, and ten years later it still accompanied them. As in the last case, there was a family history of headaches, of gout, and also of rheumatism. The day before one of her headaches, she always felt particularly well; although easily tired as a rule, she then felt as if she could walk any distance. (This antecedent is often described by the sufferers from migraine.) Her headaches began suddenly, without any preceding sensory symptoms, with pain at the back of one eye, either right or left. It spread through that side of the head and then invaded the other side, without ceasing on that first affected. When at its height she often had a strange feeling that she was going to say or do something odd, and immediately began to ramble in her talk. This wandering state often continued for an hour or more ; sometimes it lasted only three or four minutes. It passed away with a dazed feeling, not with any sense of faintness. Afterwards she remembered the strange initial sensation that she was going to talk nonsense, but absolutely nothing of the period of delirium.

Epilepsy from Migraine.—Such delirium as I have described may not only resemble the post-epileptic state in its character, but its nature may, perhaps, be similar. We ascribe the automatic action, and also the hysteroid state met with after slight epileptic seizures, to transient loss of control of the structures involved. We can

conceive that the intense pain may produce a similar effect, inhibiting the controlling centres. It can only be a conception, because we know nothing of the actual mechanism of the pain. But the influence on the cerebral functions is important for our present subject, because it may be more profound. We have seen, from the last case, that the mental aberration may be brief. Such a condition, if more profound, might produce a loss of consciousness very similar in aspect to that of epilepsy, as in the case I am about to mention.

A married woman, aged 47 years, had suffered for a long time from occasional headaches ending in vomiting. During some of these, when the pain had become very intense, she lost consciousness for a few moments, the loss being preceded by a sense of falling, attended by pallor of the face, and followed by cold perspiration. It was probably more than syncope, for consciousness returned through "dazedness," not by a sense of faintness. After a time she occasionally had a similar attack when she had no headache. Thus it seemed as if the effect of the pain had produced a tendency which became independent of its excitant. Such a case passes out of the borderland of epilepsy, over the border line.

Equally distinct was the relation of epilepsy to migraine in the case of a married woman, aged 38 years, without neurotic heredity, who had suffered from periodical headaches since the age of six. For many years they occurred at intervals of about three months, but they subsequently became more frequent. The headaches were chiefly frontal, severe, lasting all day, without any sensory prodroma. For many years, when the pain became most intense, she suddenly felt a peculiar sensation at the epigastrium, which passed up, through the chest to the head, and seemed to spread over the whole head. It continued for ten minutes, and ended as suddenly as it

began, but with an eructation of flatus. During the sensation she was unable to see, or to speak, or to understand what was said to her. She heard the words as sounds but could not tell their meaning. When the sensation ended sight returned, and her power of speech came back gradually; at first, on trying to say a word, she could not utter it correctly. This arrest of cerebral function at the height of the pain, following a sensation resembling the epigastric aura of epilepsy, is sufficiently remarkable, although consciousness was unimpaired. But it proved to be more than a resemblance. Five years before she was seen, at the age of thirty-three, this sudden state became a definite epileptic fit. When the headache reached its height she lost consciousness, and, if standing, she fell, without warning; general convulsion occurred, with tongue-biting and micturition. These fits had become her chief trouble, but they always occurred during the course of the headache. Thus the development of the epileptic attacks from the cerebral disturbance during the pain, apparently as its effect, is clear. Unfortunately, I saw the patient only once; but she was intelligent, and gave a clear account of her symptoms. The facts are in close harmony with those of the preceding cases.

Prodromal Connection.—Another remarkable illustration of a relation between migraine and epilepsy is afforded by a case in which the headaches did not cease when epilepsy set in, but became preceded by the aura of the fits. The aura was visual, and the headaches at once appropriated it. This is the only interpretation we can put upon the facts. The patient was a girl—sixteen when seen—in whose family there was a history of gout on the mother's and insanity on the father's side. From about the age of five she had been liable to periodical headaches,

general. When thirteen-and-a-half she had an epileptic fit during sleep; since then three others had occurred—two during sleep and one when awake. Since the first fit the headaches had continued, but preceded by a brief, very definite, visual spectrum, and the single epileptic attack which occurred when she was awake was preceded by precisely the same aura. The headaches had been frequent, at intervals of about six weeks, and the preceding visual spectrum was always of the same character, so that she was able to give a precise description of it. A bright object appeared a little to the right of the fixing point; it was not quite a star, although it had projections which were in constant movement. To illustrate this movement the patient moved her fingers just as cilia move. These projections were each of a different colour—yellow, blue, and red, never green. It was so bright that she called it "fiery," and behind it there was a misty dimness, not blackness. At once it began moving to the left, and when it had passed to a place about half-way from the fixing point to the left edge of the combined fields it stopped and moved to the right, passing above the fixing point, obliquely downwards to the extreme right edge of the field, "as far as she could see to the right," and she felt compelled to follow it. Then it passed again to the left the same distance as before, and again to the right, although not quite so far. The movements were repeated two or three times, and then the object suddenly disappeared, she could not say where. The duration was estimated at "a minute." It left for a time dimness of sight in the region through which it had passed, the middle and right parts of the combined field. For instance, she said that immediately after it had disappeared, if looking at a person's face who was near her, she would only be able to see the ear that was on her left side. As soon as it ceased, pain in the

head came on; sometimes it was bilateral and frontal, from temple to temple, lasting most of the day, and ending with vomiting; more often it was left-sided, and especially intense just above the eyeball, at a spot indicated as midway between the eyeball and the eyebrow. The pain here began as soon as the luminous spectrum ceased, and continued for several hours. During a year the patient was under observation she had no more fits, and the headaches became slight, but the spectrum occasionally occurred before them.

This case is specially instructive. The visual sensation resembled in its brevity an epileptic aura rather than a prodroma of migraine; it was similar also in its movement as a whole, in its change of place but not of character, and likewise in the sense of a compulsion to follow its movement across the field. The visual process seems to have been introduced into the brain disturbance by the development of epilepsy and immediately to have attached itself also to the attacks of migraine. It preceded these after the first fit, which was during sleep, and some time passed before the single attack in the day revealed its relation to the epilepsy. Although as brief before the headaches as before the fits, there was no trace of any minor attack after it, and the succeeding pain was characteristic of migraine. A focus of pain behind or above the eyeball is common in these cases, and its mystery is increased by the fact that, like the one-sided cranial pain, it is on the side opposite to the preceding peripheral disturbance. In this case the visual spectrum was in the region of the combined field subserved by the right eye. It did not invade the left portion of the fields subserved only by the left eye and the right hemisphere. This agrees with the occurrence of the localised pain on the left side.

Another instance of the prodromal connection of

epilepsy and migraine was afforded by a girl, aged 23 years, who had suffered for long from attacks of pain over one eye, lasting twelve hours and ending in sickness. There was a strong family history of epilepsy, and at twenty-two she had a severe convulsive fit, after which she vomited undigested currants. Nine months afterwards a second occurred. Since the first the headaches had almost ceased: only one attack had occurred—another instance of replacement. After the first fit she had occasionally experienced a brief dreamy feeling, with a sense of unreality, and as if sounds she heard were distant. This was attended by a peculiar sensation in the roof of the mouth and lips, a sense of tingling, of the part "going to sleep." This often recurred afterwards without the dreamy sensation, but with a cramped feeling in the lips and a transient difficulty in uttering words. Sometimes after this there was a sensation as if of impending faintness, but not actual faintness. Once there was a similar tingling in the left arm above the elbow passing down to the hand, and then felt in the mouth. After five minutes it ceased and a bad headache came on, which continued all the rest of the day, right-sided, fixing itself over the right eye.

This sensation in the lips and palate is essentially a pre-migraine symptom. Yet it was at first associated with the brief dreamy state characteristic of epilepsy, and this was confirmed by the convulsive attacks she had had. The latter were at once arrested by bromide and lactate of zinc, and the dreamy states also gradually ceased. When last seen she had no epileptic symptom for five years. But the pre-migraine sensation in the mouth continued, and after a year or so it was followed by characteristic headache. The prodroma did not occur with the early migraine but seemed to be developed by the epilepsy, and, persisting independently, to become

ultimately connected with the pain. The facts illustrate also the migrainous significance of lip and mouth tingling and the hindrance to speech that it may cause. (Compare the case described on p. 81.)

The same relation of the two diseases, the prodroma of the headaches becoming the warning of epilepsy, was presented by a man, aged 60 years, who had become liable to senile migraine at fifty-five. At intervals of a few months he experienced a *zigzag* visual spectrum, sometimes of prismatic colours, sometimes only bright light. It was described as often involving hemianopia, usually lateral, but sometimes transverse, the lower half of the field being lost. With it there was often vertical diplopia. These symptoms were often followed by severe pain in the eyeballs, but occasionally by quite sudden loss of consciousness. It was apparently epileptic, since bilateral twitching was observed; on several occasions his cheek was bitten, and once a motion was passed unconsciously. Before the onset he generally felt specially cold; nitrite of amyl failed to avert the attacks. Bromide and belladonna, with trinitrine, almost arrested the symptoms, since during a year he had only one or two trifling visual spectra, with no sequel. It was apparently a case of senile epilepsy with a migrainous aura which might have pain or convulsion as its sequel.

Elaborate Premonitory Symptoms.—The disturbance that precedes paroxysmal headaches sometimes presents a very complex character, the symptoms being unusual in their variety and severe in degree. They may then give rise to much perplexity, and their true nature may not be recognised. The following case is an instructive example of the elaboration that may be attained and of the difficulty in diagnosis that may reasonably result. It

arises from the fact that the character of the initial symptoms makes it easy to regard the pain as the secondary consequence of the disturbance, when it is really the forerunner of the headache. The case is also unusual and important in presenting motor symptoms as part of the complex prodroma. It deserves careful consideration, because no two cases of this kind are alike, and it is necessary to grasp the general principles of their diagnosis.

The patient was a married woman, aged 31 years, who had suffered since eighteen from headaches, about once a fortnight. They were very intense, limited to the right side, and ending with sickness. Three years before, at twenty-eight, a sorrow befell her, the death of her mother. Since then the headaches had changed in character, and an elaborate prodroma had preceded each. First her sight was lost, not on one side, but in front of her, where "a black curtain seemed to have dropped down, brilliant with thousands of golden points." Presently the luminous points disappeared, and were replaced by a feeling that the whole room was turning towards the right, though she was still. This was followed by a sensation that her limbs, arms and legs, and also her jaw, were being fixed—"drawn" was her expression; apparently they passed into a state of rigidity, like that of tetany in character, but not in form, since the fingers were flexed at all joints. After this had continued for "ten minutes" she became "unconscious" (apparently really so) for about a quarter of an hour, but this state was sometimes replaced by a sense of faintness, followed by general tremor, with chattering of the teeth. Then the headache came on; the pain was felt at first in both mastoid processes, and passed up to the top of the head and thence down to the throat. After about a year, it began in the ears and extended to the vertex, but then

passed to the middle of the cervical spine, where it was very intense. It always continued for several hours. In connection with the pain at the ears a curious fact was described. The day after each attack of pain she almost always had a yellowish discharge from the meatus. This is quite credible, as a "flux," the effect of the pain, analogous to the subcutaneous swelling of the forehead, etc., met with in cases of neuralgia. There was some reduction of nerve-hearing on the left side, but each membrana tympani and meatus were normal.

In spite of the unusual prodromal symptoms, they were dominated by the pain, most intense and prolonged. Although complex and strange, they were only an elaborate premonition of the pain, and the discernment of this fact and recognition of its significance afford the most important lesson the case can teach. It should prevent a misapprehension, even of the disturbance of consciousness, most rare as a part of the premonition, and so easily mistaken for a symptom of epilepsy. It should even prevent a mistake as to the nature and meaning of the motor symptoms, regarding which a word more must be said. Such contracture may be due to a state of the motor centres similar to that which, in the sensory region, causes the subjective "numbness," and surprise may reasonably be felt that it does not often accompany the sensation. Such general fixity of the limbs is a condition quite different from epileptic spasm, and should not mislead, even apart from its place in a series of functional states leading up to the headache. (Compare also the case described on p. 86.)

Conclusions.—The literature of migraine is large, but it contains little that bears on the relations to epilepsy that have been considered here. Since no novelty is

assumed, it is unnecessary to refer to the writings of others. Yet the admirable monograph of Dr. E. Liveing* should not be unmentioned. In it will be found an important account of the speculations that have been put forward regarding the nature of the process; some are opposed in character, and none touch more than the fringe of the problem involved. At present it seems more useful to gather facts than to formulate hypotheses, and especially facts on the outskirts of the affection, which connect its symptoms with those of other maladies.

The traces of a definite relation of migraine to epilepsy are slight. In extremely rare instances one affection may develop while the other goes on, and, as we have seen, the same premonitory disturbance may even be attached to each. But such cases are so rare as rather to emphasise the rule to which they form exceptions. When the exceptions are carefully examined they show that any relation to epilepsy is indirect. Epilepsy develops from migraine as an indirect consequence of the effect of the intense pain and associated cerebral disturbance. The influence of this may be traced through stupor, sleep, delirium, and loss of consciousness, to definite epileptic symptoms, as in the cases that have been considered.

The most frequent relation of migraine to epilepsy is as a source of error. Yet the differences between the two affections are definite and distinctive. Their antecedents differ. When heredity is to be traced in epilepsy the antecedents present either the same disease or insanity. These are seldom to be heard of in the case of migraine, in the more marked forms of which parental or ancestral gout is seldom absent, and generally is pronounced; and there is often also a history of severe headaches or neuralgia. However difficult it may be to understand,

* 'Megrim,' etc., London, Churchill, 1873.

observation makes it certain that the offspring of gouty parents are specially liable to the painful neuroses, and this is especially true of those who are of the female sex. Other differences have been emphasised in connection with the cases that illustrate them. The difference in the duration of the premonitory sensory symptoms is especially important. The brief warning of the epileptic fit, lasting only a few seconds, is not met with before pure migraine; to this the deliberate disturbance, lasting ten to twenty or thirty minutes, is confined. Rarely it is over in five minutes, but in such cases the same prodroma on other occasions endures for a much longer time, and the variations indicate its nature. The character of the fore-running disturbance is generally also distinctive. The zone of tingling in the arm, slowly passing up the limb, is never described in epilepsy; of which any clonic spasm is distinctive, but unknown in migraine. The visual symptoms which so often precede migraine similarly differ in duration from those that constitute the warning in epilepsy, and they usually differ also in character. Dimness or loss of sight is general in epilepsy, and is momentary; in migraine it is blotchy and irregular, or one-sided and prolonged. A more definite sensation has a mechanical elaboration, and consists of an angled line, simple or multiple, straight, curved, or expanding, within which vision is dim. A stellate body, still or moving, may occur in either affection, but the simple flash of light, or multiple momentary bright "stars," so common in epilepsy, are not known in migraine. In this, also, we never meet with the elaborate psycho-visual sensation—a face, a figure, a scene, or the like, which are not rare in epilepsy. The contrast is curious between this form of elaboration and that which is so common in migraine, which I have called "mechanical"; it suggests that the latter is due to a disturbance in a region of the cortex

that is at a lower level of function than that in which the epileptic process occurs, or that the mode of disturbance is quite different.

The special characters of the pain are also important. The severe degree and long duration common in migraine are never met with after an epileptic attack of minor character, with which alone the symptoms of migraine can be confused, nor is severe headache, even after a convulsion, one-sided. Vomiting occurs soon after the fit in epilepsy, not after the headache has lasted some hours, as in migraine. The occasional occurrence of prodromal symptoms during the course of the headache may give rise to error, but this should be prevented by a knowledge of the fact. The effects of the pain itself, stupor, somnolence, delirium, and loss of consciousness, also need only to be known for the due recognition of their nature and significance.

Treatment.—The cases which seem to resemble epilepsy, and are sometimes mistaken for it, need the same treatment as other more characteristic forms of migraine. Of this, an important element is sedulous care to avoid over-fatigue of mind or body, as far as the conditions of life make it possible, and also to avoid all derangement of the digestive system. Those who suffer from regular periodical attacks often find that just after a headache they can do almost anything with impunity, while towards the end of the interval of freedom the least indiscretion will bring on an attack. The early immunity is apt to beget carelessness which is carried on too long. By thus being induced, the headaches tend to become more frequent, while persevering caution gradually prolongs the intervals. The subjects of migraine have often a highly-strung nervous system, and throw themselves with undue

energy into all they do, whether work or pleasure. Even care in diet is difficult to secure, because its effect is only to postpone and not to prevent an attack. Saline aperients taken at regular intervals have a marked influence on the frequency of the headaches, and this effect, coupled with the vomiting which results from the headache, aids the popular belief that the attacks are "bilious" in nature. There may be some element of truth in the opinion; in some cases, at least, a blood-state seems gradually to develop which excites the headache. If early symptoms permit a purgative to be taken in time, the headache may be prevented in some sufferers, though not in others.

A remarkable fact regarding the typical severe cases of migraine, in which the headaches begin in or before early adult life, and present characteristic features, is the great frequency in which the sufferers present a strong gouty heredity. The fact has been already referred to. It compels the belief that migraine is a neurotic effect of the inherited influence. It is not gout, but is certainly related to it in some unknown way. The therapeutic significance of the fact is not so great as might be expected. It does not explain the beneficial influence of aperients, although in harmony with it.

The attacks of headache are often relieved by one of the coal-tar series of anodynes, phenacetine or phenazone. It is important that a full dose should be given, fifteen grains, and as early in the attack as possible. The single large dose acts with more momentum than repeated small doses. Caffeine may be combined with phenazone in the proportion of one to five, and the mixture has been termed "migrainine." That which is generally so called is a simple mixture of the agents, but a preparation made by the German chemist Höchst, and bearing the same name, is said to be a compound of the two, prepared by heating them together at a certain temperature, and certainly

seems more efficacious than the simple mixture. It is given in the same dose—fifteen grains. Bromide seldom has much influence on the pain, but often enables it to be better borne, and in some cases a large dose, forty grains, taken at the beginning of the premonitory sensory symptoms, may cause an attack to abort. Indian hemp may be given with it, or hyoscine, seven minims of a solution of 1 in 1000. It is only in attacks of extreme severity, in which the cerebral disturbance amounts to delirium, that an injection of morphia is sometimes necessary, and, in the occasional exigency, is unobjectionable. If the pain culminates in convulsive symptoms, a full dose of bromide should be given at the onset of the attack.

Much good is done in many cases by regular treatment during the intervals between the attacks of pain. In the typical form of migraine, with pallor and coldness in the early stage of the headache, no agent seems so frequently useful as trinitrine, given regularly in a dose of $\frac{1}{150}$ to $\frac{1}{100}$ or $\frac{1}{80}$ grain, twice or three times a day. The liq. trinitrini is convenient and trustworthy, provided the mixture is not kept more than ten or fourteen days. Other details are given in the section on the treatment of vaso-vagal attacks (p. 37), and need not be here repeated. Strychnine may be usefully given with it, and a small dose of the tincture of gelsemium. It has to be continued regularly, if it is effective. When trinitrine fails, bromide may be tried, 15 or 20 grains twice daily, combined with small doses (four grains) of phenazone, or with Indian hemp. Any other agents may be combined that are indicated by the constitutional state.

CHAPTER VI.

SOME SLEEP SYMPTOMS.

THE subject of the derangement of sleep is far too large to permit any attempt to consider systematically its various features. But the cases in the border-land of epilepsy include some that are related to sleep, and should be described to complete the survey of the chief provinces. They are of interest also in connection with the process of sleep itself, although our ignorance of its nature is still too great to make even speculation profitable.

We know that sleep is a state of rest, during which the highest centres, subserving consciousness, are functionally separated from the lower centres, motor and sensory. During this state of rest there is a renewal of nutrition, which has been impaired during activity beyond the restorative capacity of the immediate metabolic changes. But rest of the nerve structures is not complete inactivity; this is probably incompatible with any molecular change, even restorative. The renewal entails some activity, without which it could not occur. This gentle function seems to be in ordered form, such as that which constitutes a dream. Many familiar facts suggest that the apparently dreamless sleep is only sleep in which the activity of the cortex is so absolutely isolated that the dreams are not afterwards recalled. The separation of the centres in sleep varies in degree, and therefore in effect, in a way very difficult to understand, but prolific in results.

In the lower centres of the brain, also, rest cannot involve inactivity. The state of the receptive centres during restorative rest must also involve persistent activity, since impulses are ever reaching them from the periphery. In the efferent centres the state must be the same. This is indeed involved in their mutual relation, through which reflex action may occur during even sound sleep, and the activity on the motor side is shown by the maintenance of muscular tone and other processes.

In normal sleep this gentle action of the lower centres has no effect upon those of higher function. During the waking state the latter exert control on some, at least, of those below them, and the absence of such control in sleep might be expected to allow insubordinate action to occur. In perfectly normal conditions this is unknown, but it does often occur in abnormal conditions of the nervous system. A sudden activity in the lower centres may renew the connection and arrest commencing sleep.

Transitional Disturbance.—Sudden activity of the lower centres is especially prone to occur during the process of "going to sleep." It is a state for which we have no single word analogous to that for the change which ends sleep, "awakening." The word "sleepening" would be convenient, or it may be termed "somnolescence." It is easy to understand that this transition, which we must conceive as a process of functional separation, should vary in different parts of the brain in which it occurs, and that this inequality should entail opportunities for abnormal action. When sleep is fully established, the lower centres subside to a more tranquil state; moreover, the passage to sleep varies in duration in different persons and in different states of the nerve centres. It is longer when there has been mental excitement, shorter after great physical

fatigue. The more quickly sleep comes on, the less is the opportunity for disturbance; those who go to sleep slowly are the chief sufferers. The symptoms deserve consideration, because, in the transition state, epileptic attacks are prone to occur in some patients, although the tendency is greater in the wakening than in the sleepening transition.

A familiar symptom is the sudden start that so often rouses an incipient sleeper. It is an insubordinate action of the motor centres, occurring during the gradual withdrawal of the higher control. It may be a start of one or both legs, or of the trunk, less often of the arms. It restores the connection with the higher centres, apparently through the sensory impression it produces. When the start occurs again and again as the sleepening process is repeated, and the disturbance is a nightly event, the hindrance to sleep may become grave.

The sensory centres may be disturbed in a similar way, alone or in association with those for motion. A man, aged 60 years, when "dropping off to sleep" was often roused by a sensation that seemed to dart through his body and was followed by a start of some part. But such sensory symptoms are more often related to the special senses, and especially to hearing. A sudden loud noise disturbs commencing sleep. Some condition must determine the action of the auditory centres, and although many who suffer thus present no indication of aural disease, in others there is definite labyrinthine disturbance. A man with chronic aural vertigo was often roused by a general start, and sometimes by a sudden "crash," which he compared to an explosion within his head. Another, without labyrinthine symptoms, was similarly roused by a "clang," as if a piece of metal was struck within his head. Sometimes a sudden flash of light occurs as sleep is coming on, usually early in the process of transition.

Such sensory disturbance may be more elaborate, and may then arouse a suspicion of epilepsy. A man, aged 34 years, had been disturbed each night for several years by a sensation, either auditory or visual. The former was a "crash," seeming, as usual, within the head, but a curious feature was that he was distinctly aroused before the sound. For a moment before it occurred he seemed to be awake, but to be unable to move or speak; as he described it "in a state of paralysing tension," which was ended by the crash. The visual symptoms which sometimes replaced the sound, were sudden sparkling lights, reddish or white, attended by a sensation of having been struck a blow on the eyes, which seemed to cause the lights. Sometimes the sound was accompanied by a sensation of being lifted up and dashed on the floor, a distinctly vertiginous sensation, although he had no aural symptoms. The patient was certain that the disturbance was always on first going to sleep. The imperfect awakening before the sound is similar to that which was presented by the case described at p. 31, and indicates that the sensory connection with the higher centres may be first alone established, and that a strong impression may complete the waking. The sense of an adequate cause for the feeling, apart from the sensation itself, is analogous to the impression of a blow in vertigo. It is a curious example of what may be termed "retro-impressions," a sensation entailing an impression of its cause. The patient had tried many agents with little result, bromide, digitalis, phosphorus, etc.; the most effective was sixty minims of paraldehyde. But he had discovered that the attacks could be arrested by cold water to his face, when an attack was deliberate enough to permit this.

The opposite transition, from the sleeping to the waking state, is also a condition in which disturbance of the lower centres may occur, although more rarely. We have seen

this in connection with aural vertigo, and it is important because the state of awakening is one in which epileptic attacks are prone to occur in some subjects, and then are peculiarly difficult to influence. Other forms of disturbance are also met with in those whose nervous system is unstable. One woman, when sleep was ceasing, was sometimes roused by a sudden sensation passing from the legs to the head, which was followed by a feeling of thoracic distress, so intense that she fancied she was dying. It lasted, in extreme degree, for a few seconds only, but passed away slowly. If spoken to in this condition she heard and replied correctly, but could never afterwards recall what had been said. She had no other epileptic symptom, and the thoracic distress suggests a border-land attack, allied to those described as "vagal."

Night-Terrors.—This symptom is familiar in childhood, but the fact is less known that it may persist to adult life. It is probably always connected with some alarming dream; although this can seldom be recalled in childhood, it is sometimes remembered in later years. The emotional disturbance half wakes the sleeper, and its momentum excites intense manifestations of alarm, too strong to be controlled, and too energetic to be at once arrested on waking. In adult life the effect may be alarming; although the cessation is more rapid, the state may occasion considerable inconvenience. A man even hesitates to sleep in the house of a friend when he knows that he may rouse the household by his alarming screams in the dead of the night. He may jump out of bed, shouting loudly, and rush frantically to the door or window. The danger is less than appears, for the activity seems at once to finish the process of awakening, which is speedily complete. In this and in the emotional manifestation, the

state differs from somnambulism, in which the quiet sleep-activity has little tendency to rouse the subject. The dreams of true night-terrors seem never to be of a character to involve danger to another person, although this may exist in some semi-somnambulistic states to be presently mentioned.

Night-terrors which persist to adult life always cease, as far as I have seen, before thirty. Their continuance after childhood is more common in males than in females, and a family tendency to it may sometimes be clear. In one instance there was such persistence in three brothers, the eldest of whom, at twenty-five, had a severe attack on his wedding night, terrifying his wife, who was ignorant of his liability.

Both night-terrors and somnambulism sometimes occur in the subjects of epilepsy, and the question arises whether they are ever of the nature of post-epileptic automatism, the sequel to a slight seizure during sleep. In the cases of the kind that I have met with, a careful investigation showed that there seemed no such connection. The association is apparently mere coincidence, unless functional instability of the brain disposes to both affections.

A married woman, aged 26 years, had suffered from night terrors from early childhood, and from seventeen to nineteen she had peculiar attacks apparently of the nature of *petit mal*. She married at twenty-three, and the night-terrors continued for two years more and then ceased. She would wake up screaming, and try to get out of bed, struggling violently if restrained. At twenty-six she had a severe epileptic fit in the day, with tongue-biting and micturition, and two others during the next six months. They ceased permanently on treatment, but she again suffered from the attacks that troubled her at seventeen. They were curiously long, lasting five minutes. If speaking,

the subject vanished from her mind, and there was a sense of strangeness; she always stopped, being conscious that if she went on speaking the words would be irrelevant. If she went on reading aloud, she uttered words different from those before her. The condition ceased as suddenly as it came on. It was a long epileptoid state.

Somnambulism.—If sleep involves a functional separation between the cortical centres, the strange phenomena of sleep-walking suggest that the separation may vary in its position. In this state, high motor centres must be in an activity similar to that of the waking state, and yet isolated from those that, in due association, subserve consciousness. But the mysteries of sleep and its varieties baffle analysis; the forms of its disturbance must be recognised when they cannot be understood. Simple somnambulism is free from emotional disturbance, and differs from night-terrors in that no related dreams can afterward be recalled. Its nearest alliance among sleep-symptoms is the common talking in sleep, although the two seldom, if ever, occur together. They have one feature in common; a sleep-talker does not answer when spoken to, though he may be awaked, and a sleep-walker, while guided by his vision, is only waked by a quite unfamiliar obstacle. In each case they are determined by previous knowledge.

Half-waking somnambulism bears some resemblance to the quiet automatic action after slight epileptic seizures, but in the latter the subject is influenced by surrounding conditions and influences. This susceptibility, which is absent in pure somnambulism, is the chief source of danger. The post-epileptic state bears more resemblance to the state of partial awakening, and so also does the con-

dition in night-terrors. There is perception of external things, but a perception that is imperfect, and may involve error, even error in action, sometimes most grave. The recognition of these differences is of considerable importance, and the condition of half-waking, with its attendant risks, is well illustrated by the following case.

A middle-aged medical practitioner had suffered from what may be called "sleep-automatonism" for ten years. Indeed, it could be traced in some degree since childhood. The facts are of special interest in connection with some mysterious railway stories. When a boy of fifteen he often went to sleep partially, not altogether. Sitting by the fire he would seem to go to sleep; he continued perfectly conscious of all that was going on around him, but could neither move nor speak, until he suddenly seemed to awake and was in usual state. There must have been a state of true sleep in part only of the brain, a condition comparable to trance and to the stage of partial waking previously noted (pp. 32 and 108). During later years there had been, as it were, a reversal of the condition. During complete sleep he passed into a half waking stage, in which he had full motor power but imperfect perception. "In this he acts under some impulse, which he can often remember afterwards. He often gets up and goes to the window and opens it, but never tries to get out." Once he dragged his wife out of bed and then dragged her in again. Shortly before I saw him he had gone to sleep in a train, and suddenly got up, opened the door and stood on the step, when the cold wind fully wakened him. He remembered the event afterwards, dimly, as if he had half known what he was doing, but he could not recollect any motive for opening the door. It was a half-awake proceeding, not somnambulistic, and its resemblance to some sensational incidents that have been recorded is sufficiently instructive. It does not explain

actions that need definite motive, but it is a striking illustration of the fact that, to a man who half wakes and finds himself in a railway carriage, not perceiving its motion, the obvious action is to get out. It is curious that in this patient bromide did not prevent the sleep-actions, but did prevent all recollection of any preceding idea or motive.

Narcolepsy.—The term "narcolepsy" has been very loosely used. It was introduced by Gelineau in 1880* as a designation for attacks of sudden, brief, irresistible sleep. His patient was a man, aged 38 years, whose sleep attacks commenced at 36 and lasted from one to five minutes. They occurred under any circumstances. There was never passage of urine or fæces. He is said to have had sometimes as many as 200 a day. Otherwise his condition was normal. No treatment had any influence upon the malady.

Cases to which the term can be properly applied are extremely rare. A typical example is that of a girl, without neurotic heredity, who was twenty-two when first seen; she had suffered, since the age of sixteen, from peculiar brief attacks of sleep. No cause for their commencement could be ascertained, except the scarcely adequate influence of early rising. One to three attacks usually occurred on successive days, and then an interval of one to two months elapsed before there were others. No exciting cause for them could be found. They occurred either in the morning or in the afternoon. Each lasted five minutes or longer, seldom a quarter of an hour.

An attack began with sudden yawning and great heaviness of the eyes, so that she found it impossible to keep them open; in a minute or two she was asleep and dream-

* Gelineau, 'Gaz. des Hôp.,' 1880, Nos. 79 and 80.

ing vividly. Sometimes the dreams were about familiar things or persons, sometimes about quite strange subjects, but beyond this general impression their details could never be recalled. Often she talked to herself during the sleep, and even seemed to rouse herself by doing so, waking in the middle of a sentence. She could generally be easily awakened by being spoken to, but she had to make a considerable effort to rouse herself completely, and often went off to sleep again a second time. If she at once yielded to the tendency to sleep when it was first felt, the sleep did not last more than about five minutes, but if she struggled against it, striving to keep awake, sleep always conquered her in a short time, and then lasted longer, ten or fifteen minutes. She was seen occasionally for a year, and had a few attacks, more prolonged. In one which lasted an hour some darkness under the eyes was noticed, but otherwise her colour never changed during an attack. Once a series lasted most of the day. Her sleep at night was always good, and her general health was perfect. No headache followed the morbid sleep.

Various medicines were tried, but only one exerted an influence on the attacks—citrate of caffein. Under this she was free for several months, but they recurred when it was omitted, and then its effect was slighter. When last seen, the attacks still occurred, although at longer intervals.

In the case just described the attacks of sleep had no apparent resemblance to epilepsy, except in the general course of the affection. In another patient similar attacks of paroxysmal sleep were associated with other seizures distinctly epileptic. She was a girl, aged 14 years, with a family history of epilepsy, and had suffered for four months from attacks clearly of that nature. Sudden loss of consciousness was attended by general rigidity, both brief, and followed by sleep. But she had also, during the same time, attacks of sudden pure sleep. A sense of

lassitude and *malaise* impelled her to lie down, and she was at once soundly asleep. The sleep lasted about two hours and she waked up well. There was no indication of any minor epileptic seizure before these attacks of sleep, though evidence of them was carefully searched for. Such sleep does not follow minor attacks too slight to be obtrusive. Another fact in harmony with the difference in nature of the two forms of attack is that when bromide was given regularly the epileptic seizures ceased, but the attacks of sleep went on unchanged.

The features presented by these cases, especially by the brief periods of true sleep, beginning with rapidly deepening "sleepiness," are characteristic of the affection to which the term was first applied, and to which it should be confined. Cases have since been described under the same name which differ much in character. That of a boy, aged 18 years, described by Foot* in 1887, was apparently similar to Gelineau's, but his somnolent attacks were said not to have involved loss of consciousness. A case described by Caton in 1889† was one of continuous sleep, except during active exercise. Continuous sleep has been called by the same name by other writers, but certainly should have another designation. The term "sleeping-sickness" has been allotted to the African lethargy which Nicolas described‡ in 1880 under the designation "Somnosis," a word now disused, which, if worth reviving, might fitly be applied to similar continuous somnolence of different nature.§

Most examples of so-called "narcolepsy" described by recent writers have occurred in hysterical subjects, some-

* Foot, Acad. Med., Ireland, 'Lancet,' i, 1887, p. 25.

† Caton, Clin. Soc., February 8th, 1889.

‡ Nicolas, 'Comptes Rend. Acad. de Science,' May 10th, 1880.

§ Gelineau quotes as similar a case described by Caffe in 1862, but in this also the sleep was continuous. When roused, the patient (a man, aged 47 years) at once went to sleep again.

times after hysteroid convulsion, sometimes independently. Among the latter are cases in which the sleep attacks endured for hours, days, or weeks, and this condition was apparently such as in England has been termed "trance." To this, and also to post-convulsive sleep, the term is certainly inapplicable.

This brings before us another source of confusion—the danger of mistaking attacks of minor epilepsy for true sleep attacks. A patient with minor epilepsy, and sometimes with attacks that are more severe, who is aware of the commencing loss of consciousness, often describes his sensation as that of "going to sleep." But this is no evidence of the nature of the attack. It is common in unquestionable epilepsy. A recent writer on narcolepsy, Friedmann, described fifteen personally observed cases. From my own experience I should say that, on an average, before fifteen cases of narcolepsy were met with, 30,000 cases of epilepsy would come under observation! Most of the cases were probably minor epilepsy. This is supported by the statement that the usual duration of the attack was fifteen to twenty seconds, and that consciousness was often not completely lost.

Attacks to which the term "narcolepsy" is strictly applicable are thus very rare, and it is important that its use should be restricted to the cases in which definite brief sleep interrupts a normal state. It should not be applied to the cases in which a sleep state is interrupted only when the sufferer is roused. The discarded term "somnosis" emphasises the distinction from true narcolepsy.

Treatment.—The varied sleep symptoms differ much in their amenability to treatment. From the meagre facts regarding true narcolepsy that are at present avail-

able it is evident that effective treatment has yet to be discovered. The most important lesson they convey is that no statements are to be trusted that are not based on genuine cases of the disease, and these are sufficiently rare to explain our lack of definite knowledge.

The transitional disturbance that occurs on passing from the waking to the sleeping state, somnolescent disturbance, is often obstinate, whatever its form; especially the symptoms that are of motor nature; the starts, for instance, are often uninfluenced by bromide, which might be expected to have a sure effect. This agent seems to act upon the higher rather that the lower centres, and the disturbance of the latter, which occurs during the withdrawal of the higher influence, may go on unchecked by it. Some other hypnotics, which promote the onset of sleep, have more effect, especially hyoscine, in doses of about $\frac{1}{150}$ grain. Sulphonal and trional have less influence, but paraldehyde is sometimes successful. The object of these agents is to break the spell of habitual tendency, and when this is achieved they should be gradually discontinued. Whatever is employed must be given at least an hour before the time at which it will be needed, so that its influence may be fully exerted when somnolescence comes on.

In all forms of disturbance, whether initial or during established sleep, care should be taken to avoid evening mental excitement, the effects of which often continue throughout the night, and also to avoid all causes of gastric disorder. The relation of the stomach to sleep is familiar and mysterious. We do not know whether it is due to derangement of the gastric nerves, influencing the state of the brain, or to some product of imperfect digestion acting through the blood. The fact that an evening meal, which is difficult of digestion, may have a pronounced effect, suggests that the disturbance is through

the nervous system, although the precise mechanism is beyond our present discernment. Intestinal derangement is also effective.

Night-terrors are usually prevented by an evening dose of bromide, but this has much less influence on sleep-walking, which seems to occur in very profound sleep, and we do not know that any agent will prevent it. Any disturbance that seems to be related to a deep degree of sleep is difficult to influence, because it is not easy to make sleep lighter without rendering it unrestful. The railway incident I just mentioned shows the slight effect produced by bromide in preventing the half-waking, which must, however, be carefully distinguished from true somnambulism. The object to be attained by such agents is to try to change the precise condition of sleep, and in this the results of observation alone afford a secure guide. Besides bromide, hyoscine, and trional, the phenacetin series and caffein may be tried, and with one or the other the desired result may usually be obtained, but it must be remembered that small doses are often the most effective. Sleep may also be improved, in slight degree, by massage, immediately after retiring to bed.

A condition of defective strength underlies many of these forms of sleep disturbance, and hence nervine tonics are useful in addition to the special measures. Exercise should be carefully regulated. The influence of mountain air and of sea air on the process of sleep, is often marked and important, but it may be opposite in different persons, and here also experience must be the guide.

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